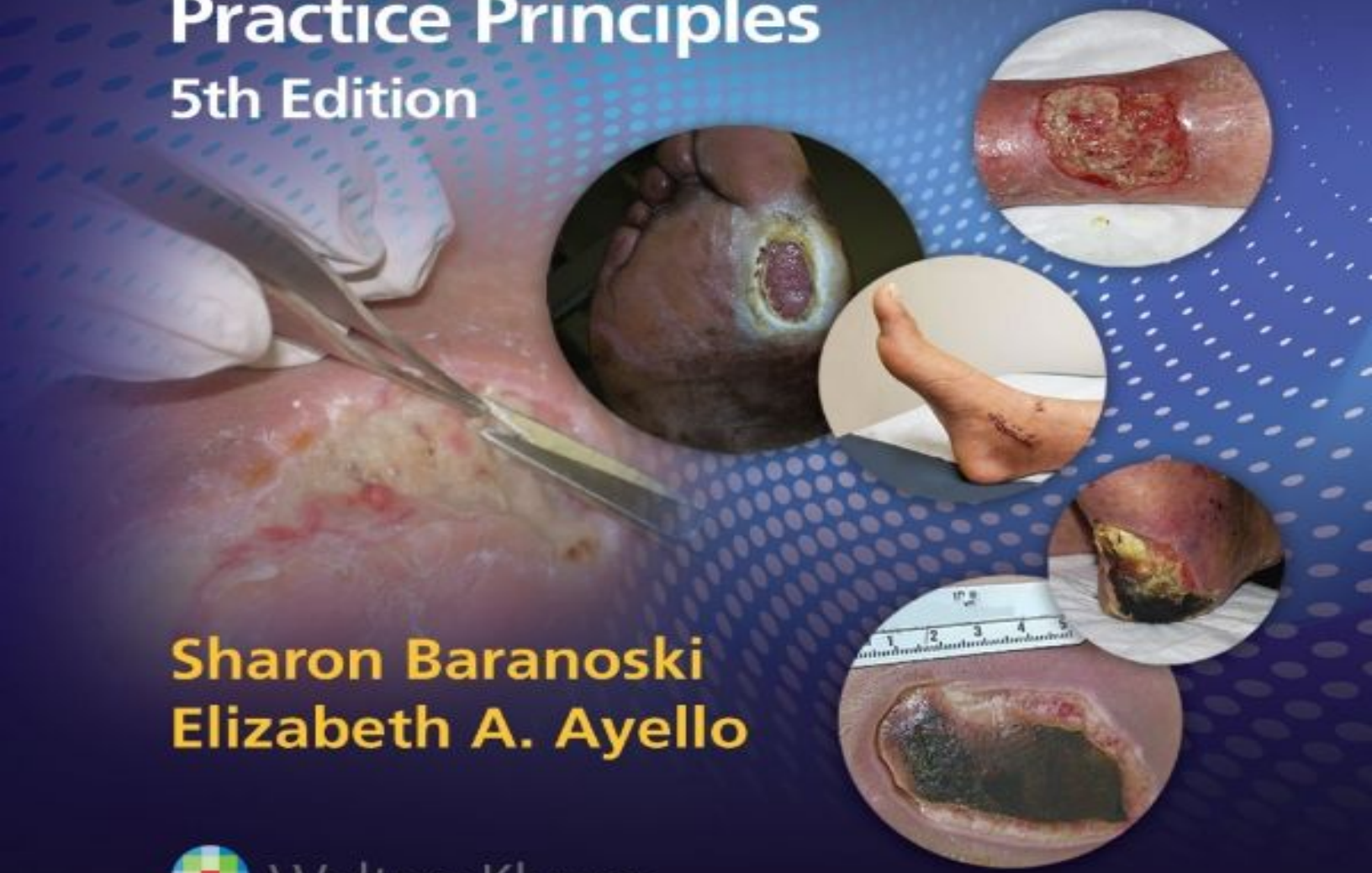
Wound Care Essentials

Practice Principles

5th Edition



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Wolters Kluwer

Wound Care Essentials
Practice Principles

Fifth Edition

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Marketing Manager: Linda Wetmore
Prepress Vendor: SPi Global

Fifth Edition

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9 8 7 6 5 4 3 2 1

Printed in China

Cataloging-in-Publication Data available on request from the Publisher

ISBN: 978-1-9751-2888-3

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Dedicaton

To my colleagues—Over the course of my nursing career, I‘ve had the privilege of working with and interacting with the best and the brightest colleagues in wound care. It has been my honor to be involved in educating, influencing, and shaping the world of wound care. It is exciting that our book will debut in 2020 the “The Year of the Nurse.”

To my family—Years have been very good to us. Our families have grown, dealt with sadness, and great change BUT we’ve had the strength to continue and always remember “family first.”

To my much loved, children and grandchildren—You’ve been the best part of my life. The laughter, smiles, tears, and “out of the mouth of babes” will stay with me forever. Love you, Madison, Lexi, Brek, Lanie, Brooklyn, Morgan, Alia, Tyler, and Taryn. To Jim, Deborah, Jeffrey, JR, and Carissa, thank you for giving me my most beautiful gifts, my grandchildren. May your lives all be filled with new adventures and exciting new chapters.

To my granddaughter and future nurse, Madison—May your career bring you as much joy and happiness as mine did. May you always remember, there is a person attached to every wound.

To my husband, Jim—Thank you for accepting me and supporting my drive, ambition, and career: you are and always will be my perfect partner, husband, father, and grandfather.

With Love, Sharon/Mom and Gramma

“**Family is everything**, a la famiglia”

This is the motto of my parents, Phyllis and Tony; my brothers, Bob and Ron and their families; and of course me and my family. Now more than ever is this true especially as more and more of my loved ones become a blessed memory.

So, thank you to my families both biological, professional, friends, and patients who have shared their experiences, supported, and nurtured me through this incredible life and skin/wound care journey.

As 2020 is the international year of the nurse and the 200th anniversary of the birth of Florence Nightingale, I want to especially acknowledge all my colleagues from the professional organizations that have enriched my nursing career especially while serving in volunteer positions—National Pressure Injury Advisory Panel (NPIAP), American Professional Wound Care Association (APWCA), Wound Ostomy and Continence Society (WOCN), and the World Council of Enterostomal Therapists (WCET®). In the almost five decades of my nursing career, there are too many of you to mention individually; hopefully I have done a good job of expressing my gratitude and appreciation to you often.

To:

Mom, Dad, Bob, Ron, and their families for the food, laughs, and the smiles

Roberta, who mentored and taught this clinical nurse specialist the skill of writing and editing over 30 years ago

My **"Dream Team"** for having me as part of an extraordinary group of educators, authors, and researchers

Katie, the sister I never had, who taught me the real meaning of unconditional friendship

Sarah, whose joy of music taught me that when words fail, listen to the true meaning of healing

Wendy and Andrei, who give me hope for the future of health care and the promise of tomorrow. I am so proud of you

A. Scott, who brings the art through his paintings, guitar playing, and singing to balance my science. Thank you for your patience while I was busy with "the book." Now let's dance.

Love E.A.A.

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Preface

> ***Tomorrow is the most important thing. … It's perfect when it arrives and it puts itself in our hands. It hopes we've learned something from yesterday." John Wayne, actor***

We cannot believe that it is almost 20 years ago that we began writing our book "Wound Care Essentials: Practice Principles." It was a labor of love that we undertook in response to the many requests that our professional colleagues made to us to create a succinct book that combined the essential knowledge, synthesis of the current evidence with the expertise gained from clinical experience. We continue to be grateful to the many people who have let us know the role that our book has played in their initial and continuing professional education. It is always exciting when at a professional conference someone comes up to us showing us the well-used and dog-eared copy of their book. Using educational resources rather than having them sit on a shelf is so important. Many people have a 5-year plan, and while we were not sure back then that there would be future editions, here we are introducing you to this fifth edition.

As our book is publishing in the 200th anniversary of the birth of Florence Nightingale, a global visionary in the management of wounds, we thought it would be a good idea to use one of her important strategies which is reflection. Therefore, we have revisited a chapter we wrote in the first edition entitled "Wound care: Where we were, where we are, where we're going." Ms. Nightingale also strongly believed in evidence base practice and therefore you will find that all chapters have been thoroughly updated with the most current research available to our authors at the time of their chapter writing.

"Wound care: Where we were, where we are, where we're going."

Where we were: One obvious change is terminology. Pressure ulcers are now referred to as pressure injuries and the National Pressure Ulcer Advisory Panel (NPUAP) is now the National Pressure Injury Advisory Panel (NPIAP) along with the European Pressure Ulcer Advisory Panel (EPUAP) and Pan Pacific Pressure Injury Alliance (PPPIA) have released the third edition in November 2019 of the International Pressure Injury Clinical Practice Guideline. Documentation in the chart is now computer-based. Electric medical records are required in all healthcare settings in the United States. Gone are the days (thankfully) of attempting to read orders in cursive.

Where we are: Clinically, the exact turning interval for repositioning people to offload pressure is being researched to see if the old adage of "turn every 2 hours." Is really evidence base. National guidelines now support 4-hour intervals if on an appropriate mattress. The research team of Dr. Nancy Bergstrom continues to provide us new research and insights upon which to base prevention strategies. Some support surfaces/beds and even a wearable device can alert the staff that a patient is not moving and needs to be turned and repositioned. In this edition of our book, you will note that we have included extended information about device-related pressure injuries (MRPI) and mucosal injuries, etiologies of pressure injuries that were not emphasized back then. The newly updated and validated Braden QD risk assessment scale for pediatrics now includes medical devices as one of the factors to be assessed.

Wound care education has changed with shorter more frequent presentations and more and more online and distant education offerings available. Education has become more interactive, case based, global and interprofessional focused. Since many of our devoted readers have told us how much you like the "Show what you know questions," we have increased the number of questions in some chapters as well as the number of photos in our popular Wound Gallery.

Suggested in our original book that minimally invasive surgery would reduce the number of acute incision wounds—which is now true. Robots are part of the surgical team process as new techniques

are implemented; some of which have resulted in getting patients out of patients sooner (within hours of surgery). It will be interesting to see the impact (if any) on post-op patient care needs and/or complications. Truly new ways of thinking have to replace old practices.

Newer dressings continue to emerge and those with less aggressive adhesion have greatly reduced the pain of dressing removal and increased patient comfort. Who ever thought we could “peak” under a dressing to see the wound and then the dressing could be put back in place? Dressings are an important aspect of wound management, but we must also remember that other supportive measures like nutrition, antiseptics, and systemic antibiotics need to be given equal attention.

Technology has come a long way in the past 20 years. We predicted handheld scanning devices to detect stage 1 pressure injuries, and portable ultrasound equipment is now available for such assessments. A new device provides clinicians with visual pictures of the blood flow in the wound after HBOT treatments providing a reliable guide to assess the effectiveness of the treatment. Low cost infrared thermography is now used for early detection of Charcot foot and wound infection in persons with diabetic foot ulcers.

Where we’re going:

Research may support tissue engineering techniques like stem cells and gene therapy for achieving wound closure. Stem cell-based skin engineering and gene recombination represents an alternative tool we hope to see in our future. Of course, our hope is that with newer prevention options, there will be less wounds that occur and thus less need for newer and advanced treatment options.

Technology will continue to evolve with ever-changing new noninvasive devices to better our assessment skills. So that clinicians can have more time to care for patients rather than spending so much of their time writing care notes, we are still hoping that technology can streamline this process and be more time efficient. We are still wishing that what we asked for in the first edition will come true—that clinicians will speak into their work badge

and that then this automatically sends their documentation notes to the patient's electronic medical record. Public policy and reimbursement have and will continue to impact practice. Health economics may govern how we practice, where we practice, and with whom we practice.

What has not changed is the caring for the whole person who has a skin or wound problem. We hope that never will. As Nightingale said, "*To our beginners, good courage, to our dear old workers, peace, fresh courage too, perseverance: for to persevere at the end is as difficult & needs yet better energy than to begin new work*."

To our new colleagues in skin and wound care, welcome, to our old colleagues, thank you for sharing your wisdom, research, and knowledge with us. We deeply appreciate your being part of our skin and wound care journey.

Sharon Baranoski and Elizabeth A. Ayello

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Objectives

After completing this chapter, you'll be able to:

- describe how wounds and those afflicted by wounds are viewed
- identify the impact of quality of life on patients with wounds and their caregivers
- describe the ethical dilemmas confronted in wound care
- identify issues and challenges faced by caregivers of patients with wounds
- describe the strategies aimed at meeting the needs of patients with wounds and their caregivers.

Introduction

Wound healing involves complex biochemical and cellular events. Chronic wounds do not follow a predictable or expected healing pathway, and they may persist for months or years.[1,2] The exact mechanisms that contribute to poor wound healing remain elusive; an intricate interplay of systemic and local factors is likely involved.[3] With an aging population and increased prevalence of chronic diseases, the majority of wounds are becoming recalcitrant to healing, placing a significant burden on the health system and individuals living with wounds and their caregivers. Although complete healing may seem to be the desirable objective for most patients and clinicians, some wounds do not have the potential to heal due to factors such as inadequate vasculature, coexisting medical conditions (terminal disease, end-stage organ failure, and other life-threatening health conditions), and medications that interfere with the healing process.[4] Whether healing is achievable or not, holistic wound care should always include measures that promote comfort and dignity, relieve suffering, and improve quality of life (QoL).

Case Study

Margaret is an 86-year-old woman who resides in a long-term care facility. With progressive dementia over the last 5 years, she has become incontinent and experienced significant weight loss due to poor oral intake. Margaret developed a stage 4 pressure injury (PI) in the sacral area after a recent hospitalization for exacerbation of heart failure. She continues to exhibit symptoms of dyspnea and prefers to sit in a high Fowler position (head of bed above 45 degrees) in bed to help breathing. She gets agitated when she is repositioned, especially in a side-lying position. Her only daughter is distraught over her mother's agitation, and she wonders if the constant repositioning is necessary. During a family meeting, the daughter asked the following questions regarding her mother's care, "If this is not something she likes, are we doing the right thing for her? Is this quality of life?"

Quality of Life and Person-Centered Concerns

What is quality of life (QoL)? Generally, QoL is defined as a general perception of well-being by an individual. It is a subjective but dynamic construct that is influenced by emotions, beliefs and values, social contexts, and interpersonal relationships, which together account for its variability.[5–7] Health-related quality of life (HRQoL) is a measure intended to capture to the sense of well-being that is specifically affected by health and illness focusing on symptoms and functions, levels of impairment, disabilities, and handicaps,[8] along with other related efforts to promote health, manage disease, and prevent recurrence.[7] HRQoL refers to a subjective appraisal of life that is predicated on the meanings, purposes, expectations, demands, and priorities a person assigned to situations, taking into account of cultural norm, sociopolitical context, and value system. The concept is shaped by person–environment transactions; it is complex, fluid, dynamic involving multiple overlapping dimensions (e.g., biological and physiological factors, symptoms, functioning, general health perceptions, and overall QoL). Each component may carry more importance at a given time based on the context of health and illness. Among people with chronic wounds, there is very little dispute that their QoL is severely diminished.[8–12]

Quality of Life Instruments

There are a number of validated instruments to measure QoL. The generic instruments most commonly used are the Medical Outcomes Study Short Form-36 (SF-36) and adaptations,[13] Research and Development 36-Item Form, Sickness Impact Profile,[14] Quality of Life Ladder,[15] Barthel Index,[16] Nottingham Health Profile,[17] and EuroQol EQ-5D.[18] Specific instruments that are used to evaluate HRQoL for patients with diabetic foot ulcers (DFUs) include the Cardiff Wound Impact Schedule Diabetes,[19,20] Norfolk Quality of Life in Diabetes Peripheral Neuropathy Questionnaire,[21] Neuro-QoL,[22] Manchester–Oxford Foot Questionnaire,[23] Ferrans and Powers Quality of Life Index-Wound Version (FPQLI-WV),[24] and DFU Scale.[25] For the leg ulcer patient population, the Hyland Leg and Foot Ulcer Questionnaire,[26] Charing Cross Venous Leg Ulcer Questionnaire,[27] and Sheffield Preference-Based Venous Leg Ulcer 5D[28] could be considered.[29–31] Despite adequate psychometric properties, most of these instruments are designed for research purposes, and clinical utility for daily use may be questionable. HRQoL is a relative concept; personal perception of life may not reconcile with the measurement of demonstrable ability to complete certain tasks. Arguably, the preponderance of life choices may not be relevant to impoverished environments and certain cultures. In additional, lacking in most tools are items to capture and credit the impact of political economy, social justice, and equity on HRQoL. Lastly, the assumption that all subscales contribute equally to the quality of life can be misleading. It is possible that improvement in one subscale representing one aspect of HRQoL may compromise other domains of HRQoL. For instance, spending money on a new outfit may improve psychological well-being but put undue stress on marital relationship.

Quality of Life and Chronic Wounds

The three major chronic wound types are pressure injuries (PIs), DFUs, and venous leg ulcers. A PI is an area of skin breakdown due to prolonged exposure to pressure and shear leading to tissue ischemia and cell death. Despite intensive efforts to prevent their occurrence, PIs remain a significant problem across the continuum of healthcare services; prevalence and incidence estimates range from 0.32% to over 47.4% depending on the setting of care, geographical locations, and methods to collect data.[32–40] PIs are linked to a number of adverse patient outcomes including prolonged hospital stay, decline in physical functioning, and death. In fact, patients with a PI have been reported to have a 3.6-fold increased risk of dying within 21 months, as compared with those without a PI.[41]

Gorecki and colleagues[41] reviewed and summarized 31 studies (10 qualitative and 21 quantitative) that examined issues related to QoL in people with PIs. Common concerns and salient issues were synthesized and categorized into the following themes:

1. Physical restrictions resulting in lifestyle changes and the need for environmental adaptations
2. Social isolation and restricted social life
3. Negative emotions and psychological responses to changes in body image, self-concept, and loss of independence
4. PI symptoms: management of pain, odor, and wound exudate
5. Health deterioration caused by PI
6. Burden on others
7. Financial hardship
8. Wound dressings, treatment, and other interventions
9. Interaction with healthcare providers
10. Perception of the cause of PI
11. Need for education about PI development, treatment, and prevention

Diabetes is one of the leading chronic diseases worldwide.[42] Persons with diabetes have a 25% lifetime risk of developing foot ulcers that precede over 80% of lower extremity amputations in this patient population.[43–48] The 5-year mortality rates have been reported to be as high as 55% and 74% for new-onset DFUs and after amputation, respectively; the number of deaths surpasses that associated with prostate cancer, breast cancer, or Hodgkin's disease.[48–50] Individuals with unhealed DFUs share some unique challenges. Due to problems using the foot and ankle, patients with foot ulcers suffer from poor mobility limiting their ability to participate in physical activities.[51–53] Mobility issues may also interfere with their performance at work resulting in loss of employment and financial hardship. Increased dependence can lead to caregiver stress and unresolved family tension. High levels of anxiety, depression, and psychological maladjustment may affect patients' abilities to participate in self-management and foot care.[53,54]

Increasing attention is placed on stigma that is experienced and internalized by individuals through interactions with other people in the workplace, healthcare facilities, and educational institutions, even in close interpersonal relationships. Stigma is a complex social construct, and it refers to negative characteristics and stereotypes that are often experienced by individuals with diabetes.[31] Especially for people with foot ulcers, they are labeled as "noncompliant" and blamed for allowing ulcers to develop due to their lack of self-control, willpower, and competence to make healthy choices. Internalization of stigma may lead to feelings of failure, embarrassment, disempowerment, low self-efficacy, and fear of being judged and prevent people from seeking help, discussing their difficulties openly, and following treatment recommendations (such as using prescription footwear and orthotics). Yet, the impact of self-stigma on people with diabetes and their abilities to participate in self-management remains underrecognized and underexplored. It is crucial that self-stigma be addressed in this population to avoid the vicious cycle of self-stigma, demoralization, and disengagement from disease management.

It is estimated that approximately 1.5 to 3.0 per 1,000 adults in North America have active leg ulcers, and the prevalence continues to

increase due to an aging population, sedentary lifestyle, and the growing prevalence of obesity.[55–58] Chronic leg ulcers involve an array of pathologies: 60% to 70% of all cases are related to venous disease, 10% due to arterial insufficiency, and 20% to 30% due to a combination of both.[59,60] Although venous leg ulcers are more common in the elderly, 22% of individuals develop their first ulcer by age 40 and 13% before age 30, hindering their ability to work and participate in social activities.[61] To understand the experience of living with leg ulceration, Briggs et al.[62] reviewed findings from 12 qualitative studies. Results were synthesized into five categories, similar to those identified above in individuals with PIs:

1. Physical effects including pain, odor, itch, leakage, and infection
2. Understanding and learning to provide care for leg ulcers
3. The benefits and disappointment in a patient–professional relationship
4. Social, physical, and financial cost of a leg ulcer
5. Psychological impact and difficult emotions (fear, anger, anxiety)

In two other reviews examining the impact of wounds on QoL, a total of 22[29] and 24 studies[63] were identified. Both qualitative and quantitative studies were included in the reviews. Pain was identified as the most common and disabling symptom leading to problems with mobility, sleep disorders, and loss of employment. Other symptoms associated with leg ulcers, including pruritus, swelling, discharge, and odor, are equally distressing but often overlooked by caregivers. In the studies, patients discussed the impact of leg ulcerations on their ability to work, carry out housework, perform personal hygiene, and participate in social/recreational activities. Patients report feeling depressed, powerless, being controlled by the ulcer, and ashamed of their body. Efforts were taken to conceal the bandages/dressings with clothing or shoes; however, the latter were often considered less attractive than what would normally be worn. Both reviews identified the need to address patient engagement and patient knowledge deficits to promote treatment adherence.[29,63–69] However, others are critical of this approach as the challenges of living with a chronic condition are increasingly recognized as constraining patients' ability

to make informed decisions about their care. This is further compounded by the challenge patients face when expected to follow conflicting “best practice” advice from professionals dealing with different aspects of the patient’s health. Approaches wherein solutions depend on educating patient to make behavior changes have been shown to increase the potential for patients to be labeled as disruptive/noncompliant and to receive suboptimal care.

Chronic Wound–Related Quality of Life (CW-QOL) Framework

Based on analysis of the interviews from a descriptive qualitative study that was designed to explore patients' and clinicians' perceptions and experiences of wound care, common themes were identified to create a conceptual framework for the concept of QoL as it relates to patients with chronic wounds (Fig. 1-1). Included in the framework are two concentric circles and a center representing the individual coping with a chronic wound. The outer circle represents the social, political, and healthcare systems within which QoL is realized and lived. The inner circle outlines six key stressors encountered by people living with chronic wounds:

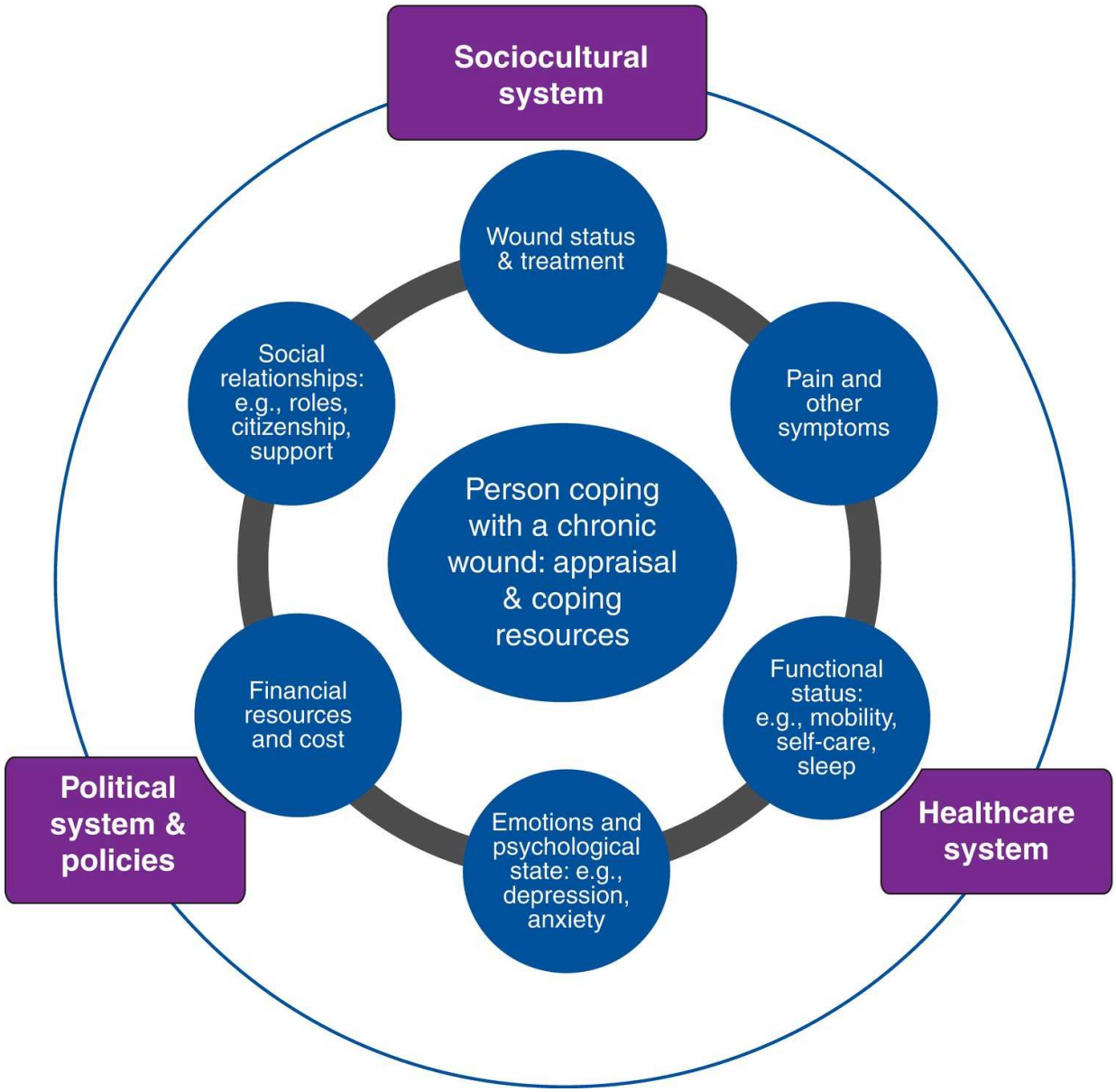

Figure 1-1 Chronic wound–related quality of life (CW-QoL) framework. (Copyright © 2014 KY Woo.)

1. Wound status and treatment
2. Pain and other wound-related symptoms
3. Function status and mobility
4. Emotions and psychological state
5. Financial resources and cost

6. Social relationships

Encumbered by increased disability and exacerbation of symptoms, patients identify a need to curtail regular recreational, social, and physical activities, and they struggle with becoming increasingly dependent on others for help. To improve patients' QoL, this paradigm places greater emphasis on the need to foster a climate that supports patient engagement as appropriate, accompanied by mindful scanning of the environment and health resource mapping. Collaborative care is the cornerstone of chronic disease self-management to help patients master problem-solving skills; the objective is for patients to experience the best possible quality of life. Individualized wound care plans that address specific patient-centered concerns are most likely to succeed and promote the best outcomes for the patient with a wound.

Person Coping with a Chronic Wound

People who are living with chronic wounds describe the experience as isolating, debilitating, depressing, and worrisome, all of which contribute to high levels of stress. Stress has a direct impact on QoL. Lazarus and Folkman[70] postulated that stress is derived from cognitive appraisals of whether a situation is perceived as a threat to one's well-being and whether coping resources that can be marshaled are sufficient to mitigate the stressor. Stress appraisal is constructed when the demands of a situation outstrip perceived coping resources.[70] While no one is immune to stress, the impact of a chronic wound on individual's perception of well-being and QoL depends on personal meanings and values that are assigned to the demands that arise from living with a chronic wound. Coping is less adaptive or effective if people lack self-esteem, motivation, and the conviction that they have the aptitude to solve a problem.[71] Woo[71] evaluated the relationship between self-perception and emotional reaction to stress in a sample of chronic wound patients. Findings suggest that people who are insecure about themselves tend to anticipate more wound-related pain and anxiety.

Chronic Stress Is Not Innocuous

Stress triggers a cascade of physiological responses featured by the activation of the hypothalamic–pituitary–adrenal axis that produces vasopressin and glucocorticoid (cortisol).[72] Vasopressin is known for its property to induce vasoconstriction that could potentially be harmful to normal wound healing by compromising the delivery of oxygen and nutrients. Cortisol attenuates the immunoinflammatory response to stress. Excessive cortisol has been demonstrated to suppress cellular differentiation and proliferation, inhibit the regeneration of endothelial cells, and delay collagen synthesis. The body of scientific evidence that substantiates the deleterious impact of protracted stress on wound healing is convincing.[73–75] In one study, Ebrecht et al.[76] evaluated healing of acute wounds created by dermal biopsy among 24 healthy volunteers. Stress levels reported by the participants via the Perceived Stress Scale were negatively correlated to wound healing rates 7 days after the biopsy ($P < 0.05$). Subjects exhibiting slow healing (below median healing rate) rated higher levels of stress during the study ($P < 0.05$) and presented higher cortisol levels 1 day after biopsy than did the fast-healing group ($P < 0.01$). Kiecolt-Glaser and colleagues[77] compared wound healing in 13 older women (mean age = 62.3 years) who were stressed from providing care for their relatives with Alzheimer disease, and 13 controls matched for age (mean age = 60.4 years). Time to achieve complete wound closure was increased by 24% or 9 days longer in the stressed caregiver versus control groups ($P < 0.05$).

Cognitive–behavioral strategies and similar psychosocial interventions are designed to help people reformulate their stress appraisal and regain a sense of control over their life's problem within an empathic and trusting milieu. Ismail et al.[78] identified 25 trials that utilized various psychological interventions (e.g., problem-solving, contract setting, goal setting, self-monitoring of behaviors) to improve diabetic self-management. Patients allocated to psychological therapies demonstrated improvement in hemoglobin A1c (12 trials, standardized effect size = −0.32; −0.57 to −0.07) and reduction of

psychological distress including depression and anxiety (5 trials, standardized effect size = −0.5; −0.95 to −0.20).

Simple problem-solving technique is easy to execute and provides a step-by-step and logical approach to help patients identify their primary problem, generate solutions, and develop feasible solutions. The key sequential steps are[79]:

1. explanation of the treatment and its rationale
2. clarification and definition of the problems
3. choice of achievable goals
4. generation of alternative solutions
5. selection of a preferred solution
6. clarification of the necessary steps to implement the solution
7. evaluation of progress.

Wound Status and Management

The trajectory for wound healing is often tortuous and unpredictable, punctuated by wound deterioration, recurrence, and other complications. Despite appropriate management and exact adherence to instructions, there is no guarantee that healing will occur. The following quotes are some of the narratives that patients voiced to convey their worry, frustration, and feeling of powerlessness.

> "The wound doctor asked me to use this dressing, but the wound is not getting better. I don't know what else to do?"[80]
> "The wound is getting bigger, and now I am getting an infection; I don't know why this is happening to me?"[80]

Even when best practice is implemented, some treatment options are not feasible and they are not conducive to enhance patients' QoL, for example, a patient with foot ulcers who cannot use a total contact cast because he needs to wear protective footwear at work and he cannot maintain his balance walking on a cast, a patient with venous leg ulcer who likes to take a shower every day to maintain personal hygiene but cannot do so because she needs to wear compression bandages, or a patient with a PI who refused an air mattress because it generates too much noise that interferes with sleep. While turning patients every 2 to 4 hours has been recommended, repositioning can be painful, especially among patients who have significant contractures, increased muscle spasticity, and spasms. Among critically ill individuals, repositioning may precipitate vascular collapse or exacerbate shortness of breath (as with, e.g., advanced heart failure).[81] According to the study of hospitalized patients with PIs,[82] it was surprising for investigators to learn that even assuming a side-lying position could be uncomfortable. Briggs and Closs[83] indicated that only 56% of patients in their study were able to tolerate full compression bandaging, with pain being the most common reason for nonadherence. Similarly, patients' adherence to wearing compression

stocking as a prophylactic measure to preempt ulceration is poor.[84] Patients should be informed of various treatment options and be empowered to be active participants in care decisions. Being an active participant involves taking part in the decision-making for the most appropriate treatment, monitoring response to treatment, and communicating concerns to healthcare providers.

When circulation is diverted from the skin to maintain hemodynamic stability and normal functioning of vital organs, skin damage is inevitable. For this reason, it is important that all involved recognize that, despite the best efforts to mitigate skin damage, in certain situations wounds are not always avoidable. Much discussion in the qualitative literature has focused on patients' lack of knowledge about how chronic wounds develop as an issue to be resolved.[29,62] As a reminder, patients, especially those dealing with the challenges of chronic disease, may lack the cognitive ability to recognize factors contributing to their chronic wounds. As self-care practices become the new norm in health care, many in this population continue to rely heavily on healthcare practitioners (HCPs) for information, advice, and support related to treatment strategies to improve their conditions.[85] By providing information about how chronic wounds are largely preventable but not always avoidable, HCP can assist patients and their families to make informed choices and reduce feelings of guilt or blame that are often associated with wound development.

Complications such as wound infection are common but upsetting. According to an analysis of an extensive database comprising approximately 185,000 patients attending family medical practitioners in Wales, 60% of patients with chronic wounds had received at least one antibiotic in a 6-month period for the treatment of wound infection.[86] Bacteria compete for nutrients and oxygen that are essential for wound healing activities, and they stimulate the overproduction of proteases leading to degradation of extracellular matrix and growth factors.[87–89] Among patients with DFUs, wound infection is one of the major risk factors that precede amputations. Surgical site infection has been linked to prolonged hospitalization and high mortality.[90,91] In fact, the mortality rate has been reported to be over 50% in patients with bacteremia secondary to uncontrolled

infection in PIs.[86,92] Receiving a diagnosis of an infection is anxiety provoking; patients often fear that infection is the beginning of a downward vicious cycle leading to hospitalization, limb amputation, and death. The need to align expectations and dispel misconceptions cannot be underestimated.

Pain and Other Symptoms

Wound-associated pain continues to be a common yet devastating symptom, often described as one of the worst aspects of living with chronic wounds.[93,94] Sleep disturbance, immobility, poor appetite, and depression are some of the consequences of unremitting pain. In an international survey of 2018 people with chronic wounds, over 60% of the respondents reported the experience of pain “quite often” and “all the time.”[95] In a survey of 287 patients with PIs, McGinnis et al.[96] reported that 75.6% of patients with stage 1 PI experienced pain with the sacrum, buttocks, and heels being the most vulnerable and painful; the mean pain intensity was 6.4 (SD 2.53) and the median 7.0 on a 10-point pain scale. Of people with venous leg ulcers, the majority experienced moderate to severe levels of pain described as aching, stabbing, sharp, tender, and tiring.[94,97,98] Pain has been documented to persist up to at least 3 months after wound closure. Contrary to the commonly held belief that most patients with DFUs do not experience pain due to neuropathy, up to 50% of patients experience painful symptoms at rest, and approximately 40% experience moderate to extreme pain climbing stairs or walking on uneven surfaces.[99,100] Patients with diabetes who report pain most or all of the time had statistically and clinically significant poorer HRQoL than those who did not report pain.[52,101,102] However, pain in diabetes is often underestimated and undertreated. The need to improve pain assessment and management is incontestable. Pharmacotherapy continues to be the mainstay for pain management. Appropriate agents are selected based on severity and specific types of pain (see Chapter 12, Pain Management and Wounds).

Pruritus

Pruritus is another frequent complaint among people with chronic wounds. Of 199 people with chronic wounds who were surveyed, Paul et al.[103,104] documented that 28.1% complained of itch. Peripheral pruritus is often triggered by pruritogens (e.g., histamine, serotonin, cytokines, and opioids), giving rise to signals that are transmitted via pain-related neuronal pathways and terminated in somatosensory cortex where the sensation of itch is perceived.[105,106] In contrast, central pruritus is associated with psychiatric disorders or damages to the nervous system mediated through opioid and serotonin receptors. For patients with wounds, itch is commonly caused by peripheral stimulation of itch receptors due to irritation of the skin and related dermatitis.[107] People with chronic wounds are exposed to a plethora of potential contact irritants accounting for approximately 80% of all cases of contact dermatitis and 20% of allergic dermatitis.[108] Excessive washing and bathing strip away surface lipid and induce dryness that can exacerbate pruritus.[109] To replenish skin moisture, humectants or lubricants should be used on a regular basis. Drug treatment with paroxetine, a selective serotonin reuptake inhibitor, and gabapentin has been shown to be beneficial in palliative care patients.[110]

Odor

Probst interviewed people with fungating breast wounds, and odor was highlighted as one of the most distressing symptoms that compromised their QoL.[111–114] Patients with DFU who were also working reported higher rates of odor, suggesting that the negative effects may be amplified in social situations.[115] Unpleasant odor and putrid discharge are associated with increased bacterial burden, particularly involving anaerobic and certain gram-negative (e.g., *Pseudomonas*) organisms. Metabolic by-products that produce this odor include volatile fatty acids (propionic, butyric, valeric, isobutyric, and isovaleric acids), volatile sulfur compounds, putrescine, and cadaverine.[105] To manage wound odor, topical charcoal-based dressings and antimicrobial and antiseptic agents are recommended.[116,117]

Exudate

Wound exudate contains endogenous protein-degrading enzymes, known as proteases or proteinases that are extremely corrosive and damaging to intact skin.[118,119] When drainage volume exceeds the fluid handling capacity of a dressing, enzyme-rich and caustic exudate may spill over the wound margins, causing maceration or tissue erosion (loss of part of the epidermis but maintaining an epidermal base) and pain.[120–122] Leakage of highly exudative wounds onto clothing, furniture, and bed linens can lead to feelings of embarrassment and inhibited sexuality and intimacy.[119,123,124] In conjunction with a thorough assessment to identify causes and remedies for excessive exudate, careful selection of discreet absorbent dressings (avoid bulky materials) will improve patients' QoL.[125,126]

Functional Status

According to the International Classification of Functioning, Disability and Health of the World Health Organization, disability refers to impairments, activity limitations, and participation restrictions because of the interaction between a health condition and other physical, social, mental, or emotional factors.[127] The majority of patients with chronic wounds suffer mobility problems, and their ability to perform activities of daily living is limited.[60,128] Activities often taken for granted by the general population, such as taking a shower, getting dressed, and even walking up the stairs, could become an enormous challenge for people with chronic wounds.[129] A study by Hyland et al.[26] revealed that of 50 patients with leg ulcers, 50% had problems getting on and off a bus and 30% had trouble climbing steps. Due to increased disability in the populations, the number of patients dependent on others for help is increasing. Requesting and receiving assistance could be a hassle and embarrassment, especially if the patient lives alone and needs regular help. Easy access to transportation and changes to living arrangements (such as widening doors for a wheelchair) will enhance individual's ability to function independently, but the effort to organize and execute the plan could be daunting.[81]

Emotional and Psychological State

People with chronic wounds tend to experience more emotional problems than people without wounds in the community and are less capable to cope with stressful events.[130] HCPs (*n* = 908), in responding to a Web-based survey, acknowledged that mental health issues are common in people with chronic wounds. Over 60% of the survey respondents indicated that between 25% and 50% of people with chronic wounds suffer from mental disorders.[131] Among all the symptoms, anxiety was rated the most common (81.5%). These results are consistent with findings from a pilot study in which over 60% of people living with chronic wounds expressed higher-than-average anxiety.[132]

Financial and Cost

Financial expenditures related to nonhealing wound care place a significant burden on the health system.[133] Patients with chronic wounds are often unemployed, marginalized, and isolated. In a study of 21 patients with DFUs by Ashford et al.[134] 79% of patients reported an inability to maintain employment secondary to decreased mobility and fear of someone inadvertently treading on their affected foot. In one study, 66 patients between 18 and 65 years old, with or at high risk to develop DFU, were asked about work-related issues. Over two-thirds (67.4%) of the respondents were not able to work due to disability, 63.6% expressed difficulty performing their duties at work that required prolonged standing or walking, and 21.2% reported leaving their jobs due to diabetic foot disease.[135]

Beyond occupational stressors and dilemmas, patients may incur additional out-of-pocket expenses for transportation, parking, telephone bills for medical follow-up, home health aide services, dressing supplies not covered by insurance, and drug costs if they have no prescription plan. Those who have no insurance but don't qualify for public assistance may be forced to tap into their savings or refinance their homes. Healthcare professionals, rather than simply dismissing patients as nonadherent, should show empathy, acknowledging access and financial hardships faced by patients, and partner with their patients in addressing these issues.[136,137]

Social Relationships and Role Function

Feeling embarrassed about the repugnant smell and fluid leakage from wounds and their bodies, people with chronic wounds may intentionally avoid social contacts and activities. Patients often feel detached and emotionally distant from their friends and families, rendering it difficult to maintain meaningful friendship and romantic relationships. Patients with chronic wounds are frequently isolated and lack social support. The concept of social support refers to an interactive process that entails perceived availability of help or support actually received. In a study of 67 patients with venous leg ulcers, Edwards and coinvestigators[138] evaluated the impact of a community model of care on QoL. Subjects were randomized to receive individual home visits from community nurses (the control group) or to pay a weekly visit to a nurse-managed leg club (the intervention group). Leg clubs offer a setting where the subjects can obtain advice/information to manage their ulcers through social interaction with expert nurses as well as with their peers. Subjects who attended the leg club expressed significant improvement in QoL ($P < 0.014$), morale ($P < 0.001$), self-esteem ($P = 0.006$), pain ($P = 0.003$), and functional ability ($P = 0.004$).

The notion that social media could be leveraged to provide virtual social support is gaining popularity. Social media encompasses a variety of platforms that provide opportunities for multiple users to exchange experiences and information and to provide support through multisensory communication. According to a 2011 survey, 80% of adult Internet users searched online and 62% used social media to obtain health information.[139] Of all the posted messages and dialogues that were abstracted from 15 Facebook groups focused on diabetes management, almost 30% of the content was related to the exchange of emotional support among members of a virtual community.[140] In one study that evaluated an online diabetes self-management program,[141] individuals randomized to the program exhibited a significant improvement in blood sugar control (A1c level),

self-reported knowledge/skill, and self-efficacy compared with those who received usual care. However, the actual participation in household activities, recreation, and exercise was not different between the two study groups. Nicholas et al.[142] designed an online module to educate and provide support to adolescents with diabetes. Participants who were randomized to the treatment group received eight online information modules and participated in peer-to-peer online dialogue that was moderated by a social worker specialized in diabetes care. Perceived social support was rated higher in the treatment group compared to the control group, but the result was not significant given the small sample size (n = 31).

Healthcare System

Navigating through the healthcare system could be extremely confusing. A trusting and therapeutic relationship between patients and their healthcare providers may serve to buffer the effects of adversity and stress. However, patients sometimes criticize about feeling rushed and spending limited time during routine visits at wound care clinic. Patients discussed the importance of having healthcare providers who care and display a genuine interest in their well-being. In their description of the key attributes of someone who cares, patients use terms like “caring,” “holistic,” “friendly,” being “vigilant,” “cheerful,” “gentle,” and “knowledgeable.” Healthcare providers should provide clear and consistent communication, to avoid confusion.

Sociocultural System

A recurring theme emerged from the literature that articulated the bleak feeling of isolation due to wound-related stigma. Given the negative image by which wounds are viewed (Table 1-1), it isn't surprising that patients with wounds are sometimes considered unattractive, imperfect, vulnerable, a nuisance to others, and, in some cases, even repulsive.[143,144] This can dramatically affect a patient's emotional response to their wound and their self-esteem. For patients who must endure the displeasing stares of others to their bandaged wounds, a wound clinic may be the only place where they can receive positive energy and reinforcement.[145,146]

Table 1-1 Emotional Impact of Wounds

In addition to the morbidity associated with wounds and the physical discomfort, wounds have an inherent emotional effect on the patient, caregivers, family, friends, and strangers the patient may encounter. Even healthcare professionals aren't immune to an emotional response to a patient's wound.

Wounds are typically perceived as:

- a betrayal of one's own body
- appalling, disgusting, and repulsive
- haunting, scary, and associated with horror movies
- nuisance, time-consuming, and costly
- smelly, dirty, and disgusting
- unpleasant and uncomfortable.
- The patient's own perception of his/her wound may include such feelings as:
- embarrassment and humiliation
- guilt and shame
- needing bandages to "hide the evidence" (i.e., of imperfection).

Political System and Policies

Health policy refers to plans, processes/structures, and actions that are established to achieve specific healthcare goals within a society. Priority setting to optimize health service delivery should include easy access to resources, appropriate funding/reimbursement mechanisms, communication strategies, and sustainable training for staff. Pressure redistribution and downloading is critical for the management of foot ulcers by removing pressure and preventing recurrent injury to the affected areas. However, these devices are expensive and may require ongoing modification by a trained professional such as a podiatrist or chiropodist. Diabetic neurotrophic ulcers may have the potential to heal but fail because downloading is not optimized. Similarly, compression therapies are used to treat venous leg ulcers. However, in most countries, there is no additional funding or reimbursement program to cover the cost for podiatric/chiropody services and the purchase of therapeutic footwear/stockings.

Conclusion

Provision of wound care requires a systematized and holistic approach to address comorbid conditions and psychosocial issues and expertise that extends beyond local wound care and dressing selection. A well-coordinated and interprofessional team approach is integral to the delivery of high-performance and evidence-based wound care services. Management of these ulcers involves a detailed examination and discussion with patients to adequately address their concerns. Although traditional educational interventions to improve knowledge are necessary, they are rarely sufficient to change behaviors. Emerging evidence highlights a need to shift the chronic disease management paradigm to focus on patient engagement and self-management (Fig. 1-2).

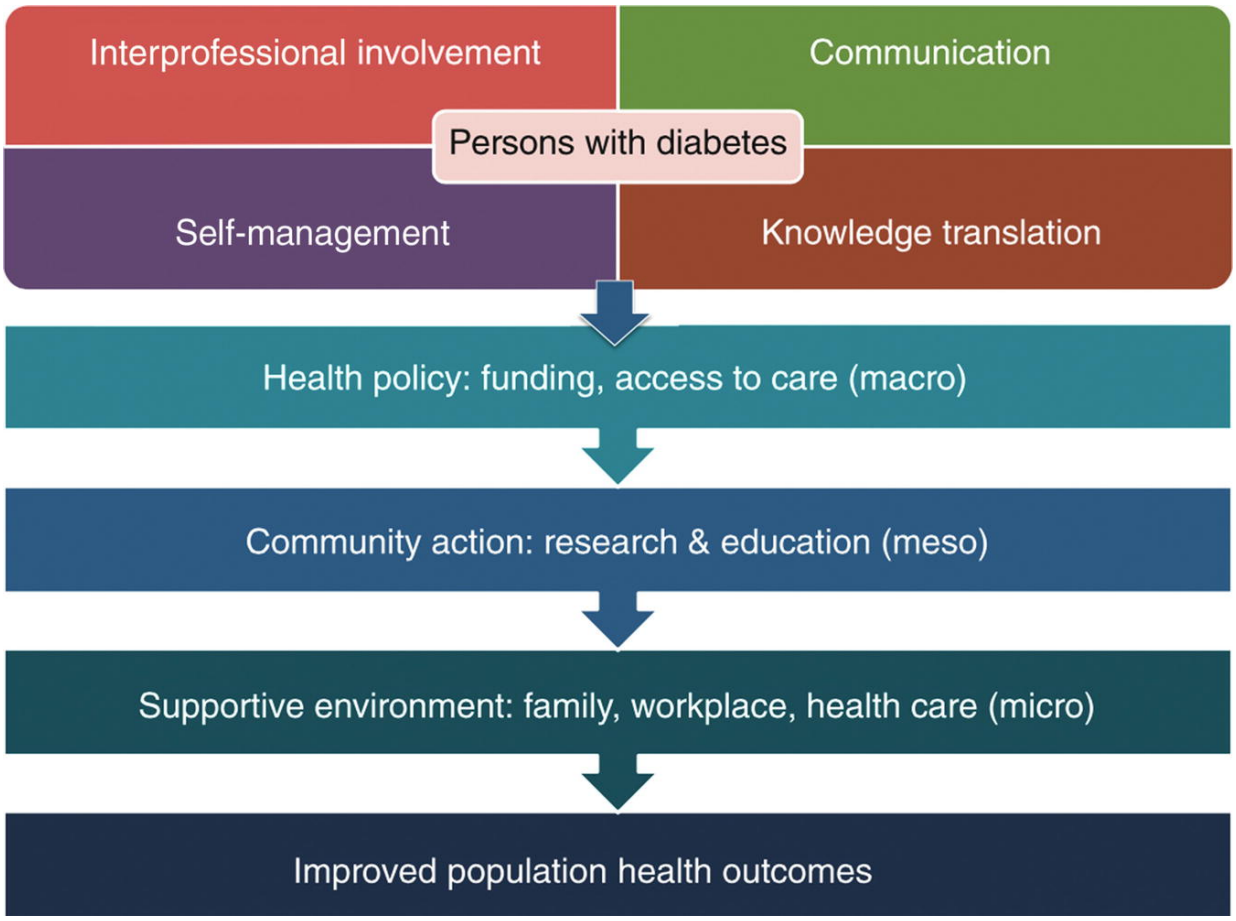

Figure 1-2 Multilevel chronic disease self-management model. (Copyright © 2014 KY Woo.)

Show What You Know

1. **Those afflicted with wounds are often viewed as:**
   A. pleasant and comfortable.
   B. pain-free.
   C. appalling and repulsive.
   D. attractive.

2. **Wound assessment is commonly lacking in the area of:**
   A. size.
   B. odor.
   C. drainage.
   D. pain.

3. **Quality-of-life treatment decisions should be based on the:**
   A. patient's perception of well-being.
   B. nurses' perceptions of well-being.
   C. family's perception of well-being.
   D. physicians' perceptions of well-being.

4. **Impact of chronic wound healing and the individual's perception of well-being includes:**
   A. stress appraisal.
   B. motivation.
   C. coping resources.
   D. all of the above.

Answers to these questions are found on page 681.

References

1. Woo, K., Ayello, E.A., Sibbald, R.G. The Edge Effect: Current Therapeutic Options to Advance the Wound Edge. Advances in Skin & Wound Care 20(2):99-117, quiz 118-9, 2007. Available at: http://www.ncbi.nlm.nih.gov/pubmed/17287621
2. Russell, D., Atkin, L., Betts, A., et al. Using a Modified Delphi Methodology to Gain Consensus on the Use of Dressings in Chronic Wounds Management. Journal of Wound Care 27(3):156-65, 2018. Available at: https://www.magonlinelibrary.com/doi/abs/10.12968/jowc.2018.27.3.156?rfr_dat=cr_pub%3Dpubmed&url_ver=Z39.88-2003&rfr_id=ori%3Arid%3Acrossref.org&journalCode=jowc
3. Martin, P., Nunan, R. Cellular and Molecular Mechanisms of Repair in Acute and Chronic Wound Healing. British Journal of Dermatology 173(2): 370-8, 2015. Available at: https://doi.org/10.1111/bjd.13954
4. Woo, K.Y. Management of Non-healable or Maintenance Wounds with Topical Povidone Iodine. International Wound Journal 11(6):622-6, 2014. Available at: https://onlinelibrary.wiley.com/doi/full/10.1111/iwj.12017
5. Zhan, L. Quality of Life: Conceptual and Measurement Issues. Journal of Advanced Nursing 17(7):795-800, 1992. Available at: http://www.ncbi.nlm.nih.gov/pubmed/1644975
6. Farquhar, M. Definitions of Quality of Life: A Taxonomy. Journal of Advanced Nursing 22(3): 502-8, 1995. Available at: http://doi.wiley.com/10.1046/j.1365-2648.1995.22030502.x
7. Price, P., Krasner, D.L. Health-Related Quality of Life and Chronic Wounds: Evidence and Implications for Practice. In D.L. Krasner, G.T. Rodeheaver, R.G. Sibbald, et al., eds. Chronic Wound Care 5: A Clinical Source Book for Health Care Professionals, Volume 1, 5th ed. Malvern, PA: HMP Communications, 2012. Available at: https://books.google.ca/books?id=TbttMAEACAAJ&printsec=frontcover&source=gbs_ge_summary_r&cad=0#v=onepage&q&f=false
8. Doward, L.C., McKenna, S.P. Defining Patient-Reported Outcomes. Value in Health 7:S4-8, 2004. Available at: https://www.sciencedirect.com/science/article/pii/S1098301510602288
9. Weldring, T., Smith, S.M.S. Patient-Reported Outcomes (PROs) and Patient-Reported Outcome Measures (PROMs). Health Services Insights 6:61-8, 2013. Available at: http://www.ncbi.nlm.nih.gov/pubmed/25114561
10. Santos, V.L.C.G., Oliveira, A.D.S., Amaral, A.F.S., et al. Quality of Life in Patients with Chronic Wounds: Magnitude of Changes and Predictive Factors. Revista da Escola de Enfermagem da U S P 51:1-8, 2017. Available at: http://www.scielo.br/scielo.php?script=sci_arttext&pid=S0080-62342017000100449&lng=en&tlng=en

11. Hopman, W.M., VanDenKerkhof, E.G., Carley, M.E., et al. Factors Associated with Health-related Quality of Life in Chronic Leg Ulceration. Quality of Life Research 23(6):1833-40, 2014. Available at: http://link.springer.com/10.1007/s11136-014-0626-7
12. Hopman, W.M., Buchanan, M., Vandenkerkhof, E.G., et al. Pain and health-related quality of life in people with chronic leg ulcers. Chronic Diseases and Injuries in Canada 33(3):167-74, 2013.
13. Rand Health Care. 36-Item Short Form Survey (SF-36). Available at: https://www.rand.org/health-care/surveys_tools/mos/36-item-short-form.html
14. Bergner, M., Bobbitt, R.A., Carter, W.B., et al. The Sickness Impact Profile: Development and Final Revision of a Health Status Measure. Medical Care 19(8):787-805, 1981.
15. Cantril, H. Pattern of Human Concerns. New Brunswick, NJ: Rutgers University Press; 1965.
16. Collin, C., Wade, D.T., Davies, S., et al. The Barthel ADL Index: A Reliability Study. International Disability Studies 10(2):61-3, 1988. Available at: http://www.tandfonline.com/doi/full/10.3109/09638288809164103
17. Hunt, S.M., McKenna, S.P., McEwen, J., et al. A Quantitative Approach to Perceived Health Status: A Validation Study. Journal of Epidemiology and Community Health 34(4):281-6, 1980. Available at: http://www.ncbi.nlm.nih.gov/pubmed/7241028
18. EuroQol Research Foundation. EQ-5D Instruments. 2017. Available at: https://euroqol.org/eq-5d-instruments/
19. Price, P., Harding, K. Cardiff Wound Impact Schedule: The Development of a Condition-specific Questionnaire to Assess Health-related Quality of Life in Patients with Chronic Wounds of the Lower Limb. International Wound Journal 1(1):10-7, 2004. Available at: http://doi.wiley.com/10.1111/j.1742-481x.2004.00007.x
20. Jaksa, P.J., Mahoney, J.L. Quality of Life in Patients with Diabetic Foot Ulcers: Validation of the Cardiff Wound Impact Schedule in a Canadian Population. International Wound Journal 7(6): 502-7, 2010. Available at: http://doi.wiley.com/10.1111/j.1742-481X.2010.00733.x
21. Vinik, E.J., Hayes, R.P., Oglesby, A., et al. The Development and Validation of the Norfolk QOL-DN, a New Measure of Patients' Perception of the Effects of Diabetes and Diabetic Neuropathy. Diabetes Technology & Therapeutics 7(3):497-508, 2005. Available at: http://www.liebertpub.com/doi/10.1089/dia.2005.7.497
22. Cella, D., Lai, J.-S., Nowinski, C.J., et al. Neuro-QOL: Brief Measures of Health-related Quality of Life for Clinical Research in Neurology. Neurology 78(23):1860-7, 2012. Available at: http://www.ncbi.nlm.nih.gov/pubmed/22573626
23. Morley, D., Jenkinson, C., Doll, H., et al. The Manchester-Oxford Foot Questionnaire (MOXFQ): Development and Validation of a Summary Index

Score. Bone & Joint Research 2(4):66-9, 2013. Available at: http://www.ncbi.nlm.nih.gov/pubmed/23673374
24. Yamada, B.F.A., Santos, V.L.C.G. Construção e Validação do Índice de Qualidade de Vida de Ferrans & Powers: Versão Feridas. Revista da Escola de Enfermagem da U S P 43(spe):1105-13, 2009. Available at: http://www.scielo.br/scielo.php?script=sci_arttext&pid=S0080-62342009000500015&lng=pt&tlng=pt
25. Bann, C.M., Fehnel, S.E., Gagnon, D.D. Development and Validation of the Diabetic Foot Ulcer Scale Short Form (DFS-SF). PharmacoEconomics 21(17): 1277-90, 2003. Available at: http://www.ncbi.nlm.nih.gov/pubmed/14986739
26. Hyland, M.E., Ley, A., Thomson, B. Quality of Life of Leg Ulcer Patients: Questionnaire and Preliminary Findings. Journal of Wound Care 3(6):294-8, 1994. Available at: http://www.magonlinelibrary.com/doi/10.12968/jowc.1994.3.6.294
27. Smith, J.J., Guest, M.G., Greenhalgh, R.M., et al. Measuring the Quality of Life in Patients with Venous Ulcers. Journal of Vascular Surgery 31(4):642-9, 2000. Available at: http://www.ncbi.nlm.nih.gov/pubmed/10753271
28. Palfreyman, S., Brazier, J. PCV80 The Development and Valuation of the Sheffield Preference Based Leg Ulcer Questionnaire (SPVU-5D). Value in Health 15(4):A126, 2012. Available at: https://linkinghub.elsevier.com/retrieve/pii/S1098301512007486
29. González-Consuegra, R.V., Verdú, J. Quality of Life in People with Venous Leg Ulcers: An Integrative Review. Journal of Advanced Nursing 67(5):926-44, 2011. Available at: http://doi.wiley.com/10.1111/j.1365-2648.2010.05568.x
30. Poku, E., Aber, A., Phillips, P., et al. Systematic Review Assessing the Measurement Properties of Patient-Reported Outcomes for Venous Leg Ulcers. BJS Open 1(5):138-47, 2017. Available at: http://doi.wiley.com/10.1002/bjs5.25
31. Hogg, F.R.A., Peach, G., Price, P., et al. Measures of Health-Related Quality of Life in Diabetes-related Foot Disease: A Systematic Review. Diabetologia 55(3):552-65, 2012. Available at: http://link.springer.com/10.1007/s00125-011-2372-5
32. VanGilder, C., MacFarlane, G.D., Harrison, P., et al. The Demographics of Suspected Deep Tissue Injury in the United States. Advances in Skin & Wound Care 23(6):254-61, 2010. Available at: http://www.ncbi.nlm.nih.gov/pubmed/20489387
33. Strazzieri-Pulido, K.C., Carol, C.V., Nogueira, P.C., et al. Pressure Injuries in Critical Patients: Incidence, Patient-Associated Factors, and Nursing Workload. Journal of Nursing Management 27(2):301-10, 2019. Available at: https://onlinelibrary.wiley.com/doi/abs/10.1111/jonm.12671
34. Kim, G., Lee, J., Kim, J., et al. Prevalence of Pressure Injuries Nationwide from 2009 to 2015: Results from the National Inpatient Sample Database in

Korea. International Journal of Environmental Research and Public Health 16(5):704, 2019. Available at: http://www.mdpi.com/1660-4601/16/5/704
35. Cowan, L.J., Ahn, H., Flores, M., et al. Pressure Ulcer Prevalence by Level of Paralysis in Patients with Spinal Cord Injury in Long-Term Care. Advances in Skin & Wound Care 32(3):122-30, 2019. Available at: http://www.ncbi.nlm.nih.gov/pubmed/30801350
36. Kirkland-Khyn, H., Teleten, O., Joseph, R., et al. A Descriptive Study of Hospital- and Community-Acquired Pressure Ulcers/Injuries. Wound Management & Prevention 65(2):14-9, 2019. Available at: http://www.ncbi.nlm.nih.gov/pubmed/30730301
37. Liu, Y., Wu, X., Ma, Y., et al. The Prevalence, Incidence, and Associated Factors of Pressure Injuries Among Immobile Inpatients: A Multicentre, Cross-Sectional, Exploratory Descriptive Study in China. International Wound Journal 16(2):459-66, 2019. Available at: https://onlinelibrary.wiley.com/doi/abs/10.1111/iwj.13054
38. Goodman, L., Khemani, E., Cacao, F., et al. A Comparison of Hospital-Acquired Pressure Injuries in Intensive Care and Non-Intensive Care Units: A Multifaceted Quality Improvement Initiative. BMJ Open Quality 7(4):e000425, 2018. Available at: http://qir.bmj.com/lookup/doi/10.1136/bmjoq-2018-000425
39. Kayser, S.A., VanGilder, C.A., Lachenbruch, C. Predictors of Superficial and Severe Hospital-acquired Pressure Injuries: A Cross-Sectional Study using the International Pressure Ulcer Prevalence™ Survey. International Journal of Nursing Studies 89:46-52, 2019. Available at: https://www.sciencedirect.com/science/article/pii/S0020748918302116?via%3Dihub
40. Sullivan, J., Woo, K. Comparing the Cumulative Incidence of Pressure Injuries Using Multilayer Foam Dressings in Seriously Ill and Frail Patients: A Quality Improvement Project. Surgical Technology International 33:53-7, 2018. Available at: http://www.ncbi.nlm.nih.gov/pubmed/30326139
41. Gorecki, C., Brown, J.M., Nelson, E.A., et al. Impact of Pressure Ulcers on Quality of Life in Older Patients: A Systematic Review. Journal of the American Geriatrics Society 57(7):1175-83, 2009. Available at: http://doi.wiley.com/10.1111/j.1532-5415.2009.02307.x
42. Bakker, K., Apelqvist, J., Schaper, N.C. Practical Guidelines on the Management and Prevention of the Diabetic Foot 2011. Diabetes/Metabolism Research and Reviews 28:225-31, 2012. Available at: http://doi.wiley.com/10.1002/dmrr.2253
43. Yazdanpanah, L., Shahbazian, H., Nazari, I., et al. Risk Factors Associated with Diabetic Foot Ulcer-free Survival in Patients with Diabetes. Diabetes and Metabolic Syndrome: Clinical Research & Reviews 12(6):1039-43, 2018. Available at: https://www.sciencedirect.com/science/article/abs/pii/S1871402118302480

44. Carro, G.V., Saurral, R., Sagüez, F.S., et al. Pie Diabético en pacientes internados en hospitales de latinoamerica. Medicina (Buenos Aires) 78:243-51, 2018. Available at: http://www.medicinabuenosaires.com/PMID/30125251.pdf
45. Maiya, A.G., Gundmi, S., Matpady, P., et al. Prevalence of Foot Complications in People with Type 2 Diabetes Mellitus: A Community-Based Survey in Rural Udupi. The International Journal of Lower Extremity Wounds 17(3):169-75, 2018. Available at: http://journals.sagepub.com/doi/10.1177/1534734618791853
46. Rigato, M., Pizzol, D., Tiago, A., et al. Characteristics, Prevalence, and Outcomes of Diabetic Foot Ulcers in Africa. A Systemic Review and Meta-analysis. Diabetes Research and Clinical Practice 142:63-73, 2018. Available at: http://www.ncbi.nlm.nih.gov/pubmed/29807105
47. Monteiro-Soares, M., Boyko, E.J., Ribeiro, J., et al. Risk Stratification Systems for Diabetic Foot Ulcers: A Systematic Review. Diabetologia 54(5):1190-9, 2011. Available at: http://link.springer.com/10.1007/s00125-010-2030-3
48. Robbins, J.M., Strauss, G., Aron, D., et al. Mortality Rates and Diabetic Foot Ulcers: Is It Time to Communicate Mortality Risk to Patients with Diabetic Foot Ulceration? Journal of the American Podiatric Medical Association 98(6):489-93, 2008. Available at: http://www.ncbi.nlm.nih.gov/pubmed/19017860
49. Shin, J.Y., Roh, S.-G., Sharaf, B., et al. Risk of Major Limb Amputation in Diabetic Foot Ulcer and Accompanying Disease: A Meta-analysis. Journal of Plastic, Reconstructive & Aesthetic Surgery 70(12): 1681-8, 2017. Available at: http://www.ncbi.nlm.nih.gov/pubmed/28865989
50. Gregg, E.W., Li, Y., Wang, J., et al. Changes in Diabetes-Related Complications in the United States, 1990–2010. The New England Journal of Medicine 370(16):1514-23, 2014. Available at: http://www.nejm.org/doi/10.1056/NEJMoa1310799
51. Ahn, J., Del Core, M.A., Wukich, D.K., et al. Scoring Mental Health Quality of Life with the SF-36 in Patients with and without Diabetes Foot Complications. The International Journal of Lower Extremity Wounds 17(1):30-5, 2018. Available at: http://journals.sagepub.com/doi/10.1177/1534734618762226
52. Wukich, D.K., Raspovic, K.M. Assessing Health-Related Quality of Life in Patients with Diabetic Foot Disease: Why Is It Important and How Can We Improve? The 2017 Roger E Pecoraro Award Lecture. Diabetes Care 41(3):391-7, 2018. Available at: http://www.ncbi.nlm.nih.gov/pubmed/29463665
53. Goodridge, D., Trepman, E., Embil, J.M. Health-Related Quality of Life in Diabetic Patients with Foot Ulcers. Journal of Wound Ostomy & Continence Nursing 32(6):368-77, 2005. Available at: http://www.ncbi.nlm.nih.gov/pubmed/16301902

54. Wu, S.-F.V., Huang, Y.-C., Lee, M.-C., et al. Self-Efficacy, Self-Care Behavior, Anxiety, and Depression in Taiwanese with Type 2 Diabetes: A Cross-Sectional. Nursing & Health Sciences 15(2):213-9, 2013. Available at: http://doi.wiley.com/10.1111/nhs.12022
55. Raeder K, Strube-Lahmann S, Müller-Werdan U, et al. [Prevalence and Influencing Factors of Chronic Wounds Among Clients of Home Care Services in Germany]. Zeitschrift für Evidenz, Fortbildung und Qualität im Gesundheitswesen 140:14-21, 2019. Available at: http://www.ncbi.nlm.nih.gov/pubmed/30777680
56. Martinengo, L., Olsson, M., Bajpai, R., et al. Prevalence of Chronic Wounds in the General Population: Systematic Review and Meta-analysis of Observational Studies. Annals of Epidemiology 29:8-15, 2019. Available at: https://www.sciencedirect.com/science/article/pii/S1047279717310311
57. Harrison, M.B., Graham, I.D., Friedberg, E., et al. Regional Planning Study. Assessing the Population with Leg and Foot Ulcers. The Canadian Nurse 97(2):18-23, 2001. Available at: http://www.ncbi.nlm.nih.gov/pubmed/11865487
58. Berenguer Pérez, M., López-Casanova, P., Sarabia Lavín, R., et al. Epidemiology of Venous Leg Ulcers in Primary Health Care: Incidence and Prevalence in a Health Centre-A Time Series Study (2010-2014). International Wound Journal 16(1):256-65, 2019. Available at: http://doi.wiley.com/10.1111/iwj.13026
59. Bergan, J.J., Schmid-Schönbein, G.W., Smith, P.D.C., et al. Chronic Venous Disease. The New England Journal of Medicine 355(5):488-98, 2006. Available at: http://www.nejm.org/doi/abs/10.1056/NEJMra055289
60. Green, J., Jester, R., McKinley, R., et al. The Impact of Chronic Venous Leg Ulcers: A Systematic Review. Journal of Wound Care 23(12):601-12, 2014. Available at: http://www.magonlinelibrary.com/doi/10.12968/jowc.2014.23.12.601
61. Brown, A. Chronic Leg Ulcers, Part 2: Do They Affect a Patient's Social Life? British Journal of Nursing 14(18):986-9, 2005. Available at: http://www.magonlinelibrary.com/doi/10.12968/bjon.2005.14.18.19888
62. Briggs, M., Flemming, K. Living with Leg Ulceration: A Synthesis of Qualitative Research. Journal of Advanced Nursing 59(4):319-28, 2007. Available at: http://doi.wiley.com/10.1111/j.1365-2648.2007.04348.x
63. Herber, O.R., Schnepp, W., Rieger, M.A. A Systematic Review on the Impact of Leg Ulceration on Patients' Quality of Life. Health and Quality of Life Outcomes 5:44, 2007.
64. Weller, C.D., Buchbinder, R., Johnston, R.V. Interventions for Helping People Adhere to Compression Treatments for Venous Leg Ulceration. Cochrane Database of Systematic Reviews (3):CD008378, 2016. Available at: http://doi.wiley.com/10.1002/14651858.CD008378.pub3

65. Jull, A., Muchoney, S., Parag, V., et al. Impact of Venous Leg Ulceration on Health-Related Quality of Life: A Synthesis of Data from Randomized Controlled Trials Compared to Population Norms. Wound Repair and Regeneration 26(2):206-12, 2018. Available at: http://doi.wiley.com/10.1111/wrr.12636
66. Joaquim, F.L., Silva, R.M.C.R.A., Pereira, E.R., et al. Application of Merleau-Pontyan Perspective on the Physical and Psychological Implications of Venous Ulcers. Revista Brasileira de Enfermagem 71(5):2469-76, 2018. Available at: http://www.scielo.br/scielo.php?script=sci_arttext&pid=S0034-71672018000502469&lng=en&tlng=en
67. Torres, S.M.S.S.O., Araújo, R.O., Costa, I.K.F., et al. Health-Related Quality of Life in Patients with Venous Leg Ulcer Treated in Primary Care in Brazil and Portugal. PLoS One 13(4):e0195990, 2018. Available at: https://dx.plos.org/10.1371/journal.pone.0195990
68. Green, J., Jester, R., McKinley, R., et al. Chronic Venous Leg Ulcer Care: Putting the Patient at the Heart of Leg Ulcer Care. Part 1: Exploring the Consultation. British Journal of Community Nursing 23(Suppl 3):S30-8, 2018. Available at: http://www.magonlinelibrary.com/doi/10.12968/bjcn.2018.23.Sup3.S30
69. Cunha, N., Campos, S., Cabete, J. Chronic Leg Ulcers Disrupt Patients' Lives: A Study of Leg Ulcer-related Life Changes and Quality of Life. British Journal of Community Nursing 22(Suppl 9):S30-7, 2017. Available at: http://www.magonlinelibrary.com/doi/10.12968/bjcn.2017.22.Sup9.S30
70. Lazarus, R.S., Folkman, S. Stress, Appraisal, and Coping. New York, NY: Springer Pub. Co, 1984:445. Available at: https://books.google.ca/books/about/Stress_appraisal_and_coping.html?id=FFBqAAAAMAAJ
71. Woo, K.Y., Bloomberg, S. Wound-Related Pain: Anxiety, Stress and Wound Healing, Vol. 6, *Clinical REVIEW*. Wounds uk. 2010. Available at: https://pdfs.semanticscholar.org/4aab/fe53f7876bcd8c90d18c49f11bdcf45b04db.pdf
72. Woo, K.Y. Exploring the Effects of Pain and Stress on Wound Healing. Advances in Skin & Wound Care 25(1):38-44, 2012.
73. Walburn, J., Vedhara, K., Hankins, M., et al. Psychological Stress and Wound Healing in Humans: A Systematic Review and Meta-Analysis. Journal of Psychosomatic Research 67(3):253-71, 2009. Available at: https://www.sciencedirect.com/science/article/pii/S0022399909001317?via%3Dihub
74. Kim, M.-H., Gorouhi, F., Ramirez, S., et al. Catecholamine Stress Alters Neutrophil Trafficking and Impairs Wound Healing by β2-Adrenergic Receptor–Mediated Upregulation of IL-6. Journal of Investigative Dermatology 134(3):809-17, 2014. Available at: https://www.sciencedirect.com/science/article/pii/S0022202X15366860

75. House, S.L. Psychological Distress and Its Impact on Wound Healing: An Integrative Review. Journal of Wound Ostomy & Continence Nursing 42(1):38-41, 2015. Available at: http://www.ncbi.nlm.nih.gov/pubmed/25549307
76. Ebrecht, M., Hextall, J., Kirtley, L.-G., et al. Perceived Stress and Cortisol Levels Predict Speed of Wound Healing in Healthy Male Adults. Psychoneuroendocrinology 29(6):798-809, 2004. Available at: https://www.sciencedirect.com/science/article/pii/S0306453003001446?via%3Dihub
77. Kiecolt-Glaser, J.K., Loving, T.J., Stowell, J.R., et al. Hostile Marital Interactions, Proinflammatory Cytokine Production, and Wound Healing. Archives of General Psychiatry 62(12):1377, 2005. Available at: http://www.ncbi.nlm.nih.gov/pubmed/16330726
78. Ismail, K., Winkley, K., Rabe-Hesketh, S. Systematic Review and Meta-Analysis of Randomised Controlled Trials of Psychological Interventions to Improve Glycaemic Control in Patients with Type 2 Diabetes. Lancet 363(9421):1589-97, 2004. Available at: http://www.ncbi.nlm.nih.gov/pubmed/15145632
79. Nezu, A.M., Nezu, C.M., D'Zurilla, T. Problem-solving Therapy: A Treatment Manual. New York, NY: Springer Publishing Company, 2013:333.
80. Available at: http://theta.utoronto.ca/content.php?pid=411861&sid=3763262
81. Woo, K., Krasner, D.L., Sibbald, R.G. Pain in People with Chronic Wounds: Clinical Strategies for Decreasing Pain and Improving Quality of Life. In D. Krasner, G. Sibbald, G. Rodeheaver, K. Woo, eds. Chronic Wound Care 5: A Clinical Source Book for Healthcare Professionals. 2012:85-96.
82. Spilsbury, K., Nelson, A., Cullum, N., et al. Pressure Ulcers and Their Treatment and Effects on Quality of *Life: Hospital Inpatient Perspectives*. Journal of Advanced Nursing 57(5):494-504, 2007. Available at: http://doi.wiley.com/10.1111/j.1365-2648.2006.04140.x
83. Briggs, M., Closs, S.J. The Prevalence of Leg Ulceration: A Review of the Literature. European Wound Management Association Journal 3(2):14-20, 2003. Available at: https://www.semanticscholar.org/paper/The-prevalence-of-leg-ulceration%3A-a-review-of-the-Dealey-Gottrup/e949b95ee03d417cbcf68f60f94c265768fdbc63
84. Nelson, E.A., Bell-Syer, S.E. Compression for Preventing Recurrence of Venous Ulcers. Cochrane Database of Systematic Reviews (9):CD002303, 2014. Available at: http://doi.wiley.com/10.1002/14651858.CD002303.pub3
85. Žulec, M., Rotar-Pavlič, D., Puharić, Z., et al. "Wounds Home Alone"—Why and How Venous Leg Ulcer Patients Self-Treat Their Ulcer: A Qualitative Content Study. International Journal of Environmental Research and Public Health 16(4):559, 2019. Available at: http://www.mdpi.com/1660-4601/16/4/559

86. Braga, I.A., Pirett, C.C.N.S., Ribas, R.M., et al. Bacterial Colonization of Pressure Ulcers: Assessment of Risk for Bloodstream Infection and Impact on Patient Outcomes. Journal of Hospital Infection 83(4):314-20, 2013. Available at: https://www.sciencedirect.com/science/article/pii/S0195670112004033?via%3Dihub
87. Woo, K. The Use of Antimicrobial Dressings in Chronic Wounds: NERDS and STONEES Principles. Surgical Technology International 20:73-82, 2010. Available at: http://surgicaltechnology.com/20-073-WH-Abstract.html
88. Omar, A., Wright, J., Schultz, G., et al. Microbial Biofilms and Chronic Wounds. Microorganisms 5(1):9, 2017. Available at: http://www.mdpi.com/2076-2607/5/1/9
89. Rajpaul, K. Biofilm in Wound Care. British Journal of Community Nursing 20(Suppl 3):S6-11, 2015. Available at: http://www.magonlinelibrary.com/doi/10.12968/bjcn.2015.20.Sup3.S6
90. Badia, J.M., Casey, A.L., Petrosillo, N., et al. Impact of Surgical Site Infection on Healthcare Costs and Patient Outcomes: A Systematic Review in Six European Countries. Journal of Hospital Infection 96(1):1-15, 2017. Available at: www.sciencedirect.com
91. Crolla, R.M.P.H., van der Laan, L., Veen, E.J., et al. Reduction of Surgical Site Infections after Implementation of a Bundle of Care. PLoS One 7(9): e44599, 2012. Available at: http://www.ncbi.nlm.nih.gov/pubmed/22962619
92. Braga, I.A., Brito, C.S., Filho, A.D., et al. Pressure Ulcer as a Reservoir of Multiresistant Gram-Negative Bacilli: Risk Factors for Colonization and Development of Bacteremia. The Brazilian Journal of Infectious Diseases 21(2):171-5, 2017. Available at: https://linkinghub.elsevier.com/retrieve/pii/S1413867016305876
93. Woo, K.Y. Chronic Wound-Associated Pain, Psychological Stress, and Wound Healing. Surgical Technology International 22:57-65, 2012. Available at: http://www.ncbi.nlm.nih.gov/pubmed/23023574
94. Nemeth, K.A., Harrison, M.B., Graham, I.D., et al. Understanding Venous Leg Ulcer Pain: Results of a Longitudinal Study. Ostomy/Wound Management 50(1):34-46, 2004. Available at: http://www.ncbi.nlm.nih.gov/pubmed/14712004
95. |Price, P.E., Fagervik-Morton, H., Mudge, E.J., et al. Dressing-Related Pain in Patients with Chronic Wounds: An International Patient Perspective. International Wound Journal 5(2):159-71, 2008. Available at: http://doi.wiley.com/10.1111/j.1742-481X.2008.00471.x
96. |McGinnis E, Briggs M, Collinson M, et al. Pressure Ulcer Related Pain in Community Populations: A Prevalence Survey. BMC Nurs 13:16, 2014.
97. Renner, R., Seikowski, K., Simon, J. Association of Pain Level, Health and Wound Status in Patients with Chronic Leg Ulcers. Acta Dermato-

Venereologica 94(1):50-3, 2014. Available at: http://www.medicaljournals.se/acta/content/?doi=10.2340/00015555-1635
98. Park, S.H., Ferreira, K., Santos, V.L. Understanding Pain and Quality of Life for Patients with Chronic Venous Ulcers. Wounds 20(11):309-20, 2008. Available at: http://www.ncbi.nlm.nih.gov/pubmed/25941830
99. Evans, A., Pinzur, M. Health-Related Quality of Life of Patients with Diabetes and Foot Ulcers. Foot & Ankle International 26(1):32-7, 2005. Available at: https://www.ncbi.nlm.nih.gov/pubmed/15680116
100. Peltier, A., Goutman, S.A., Callaghan, B.C. Painful diabetic neuropathy. BMJ 348:g1799, 2014. Available at: http://www.bmj.com/
101. Goodridge, D., Trepman, E., Sloan, J., et al. Quality of Life of Adults with Unhealed and Healed Diabetic Foot Ulcers. Foot & Ankle International 27(4):274-80, 2006. Available at: http://journals.sagepub.com/doi/10.1177/107110070602700408
102. Siersma, V., Thorsen, H., Holstein, P.E., et al. Health-Related Quality of Life Predicts Major Amputation and Death, but not Healing, in People with Diabetes Presenting with Foot Ulcers: The Eurodiale Study. Diabetes Care 37(3):694-700, 2014. Available at: http://www.ncbi.nlm.nih.gov/pubmed/24170755
103. Paul, J.C., Pieper, B., Templin, T.N. Itch: Association with Chronic Venous Disease, Pain, and Quality of Life. Journal of Wound Ostomy & Continence Nursing 38(1):46-54, 2011. Available at: http://www.ncbi.nlm.nih.gov/pubmed/21287771
104. Paul, J. A Cross-Sectional Study of Chronic Wound-related Pain and Itching. Ostomy/Wound Management 59(7):28-34, 2013. Available at: https://www.o-wm.com/article/cross-sectional-study-chronic-wound-related-pain-and-itching
105. Woo, K.Y., Sibbald, R.G. Local Wound Care for Malignant and Palliative Wounds. Advances in Skin & Wound Care 23(9):417-28, 2010. Available at: http://www.ncbi.nlm.nih.gov/pubmed/20711056
106. Parnell, L.K.S. Itching for Knowledge About Wound and Scar Pruritus. Wounds 30(1):17-26, 2018. Available at: https://www.woundsresearch.com/article/itching-knowledge-about-wound-and-scar-pruritus
107. Woo, K.Y., Sibbald, R.G. The ABCs of Skin Care for Wound Care Clinicians: Dermatitis and Eczema. Advances in Skin & Wound Care 22(5):230-6; quiz 237-8, 2009. Available at: http://www.ncbi.nlm.nih.gov/pubmed/19521290
108. Alavi, A., Sibbald, R.G., Ladizinski, B., et al. Wound-Related Allergic/Irritant Contact Dermatitis. Advances in Skin & Wound Care 29(6):278-86, 2016. Available at: http://www.ncbi.nlm.nih.gov/pubmed/27171256
109. Walters, R.M., Mao, G., Gunn, E.T., et al. Cleansing Formulations That Respect Skin Barrier Integrity. Dermatology Research and Practice 2012:495917, 2012. Available at: http://www.ncbi.nlm.nih.gov/pubmed/22927835

110. Kottner, J., Lichterfeld, A., Blume-Peytavi, U. Maintaining Skin Integrity in the Aged: A Systematic Review. British Journal of Dermatology 169(3): 528-42, 2013.
111. Probst, S., Arber, A., Faithfull, S. Malignant Fungating Wounds—The Meaning of Living in an Unbounded Body. European Journal of Oncology Nursing 17(1):38-45, 2013. Available at: https://www.sciencedirect.com/science/article/pii/S1462388912000191?via%3Dihub
112. Lima Villela-Castro, D., De Gouveia Santos, V.L.C., Woo, K. Polyhexanide Versus Metronidazole for Odor Management in Malignant (Fungating) Wounds. A Double-Blinded, Randomized, Clinical trial. Journal of Wound Ostomy & Continence Nursing 45(5):413-8, 2018. Available at: https://www.ncbi.nlm.nih.gov/pubmed/30086103
113. de Castro, D.L.V., Santos, V.L.C.G. Odor Management in Fungating Wounds with Metronidazole. Journal of Hospice & Palliative Nursing 17(1):73-9, 2015. Available at: http://content.wkhealth.com/linkback/openurl?sid=WKPTLP:landingpage&an=00129191-201502000-00013
114. Castro, D.L., Santos, V.L. Controlling Wound Odor with Metronidazole: A Systematic Review. Revista da Escola de Enfermagem da U S P 49(5):858-63, 2015. Available at: http://www.scielo.br/scielo.php?script=sci_arttext&pid=S0080-62342015000500858&lng=en&tlng=en
115. de Almeida, S.A., Salomé, G.M., Dutra, R.A.A., et al. Feelings of Powerlessness in Individuals with Either Venous or Diabetic Foot Ulcers. Journal of Tissue Viability 23(3):109-14, 2014. Available at: https://www.sciencedirect.com/science/article/pii/S0965206X14000321
116. Akhmetova, A., Saliev, T., Allan, I.U., et al. A Comprehensive Review of Topical Odor-Controlling Treatment Options for Chronic Wounds. Journal of Wound Ostomy & Continence Nursing 43(6):598-609, 2016. Available at: http://www.ncbi.nlm.nih.gov/pubmed/27684356
117. Woo, K., Hill, R., LeBlanc, K., et al. Effect of a Surfactant-Based Gel on Patient Quality of Life. Journal of Wound Care 27(10):664-78, 2018. Available at: http://www.magonlinelibrary.com/doi/10.12968/jowc.2018.27.10.664
118. Sibbald, R.G., Goodman, L., Woo, K.Y., et al. Special Considerations in Wound Bed Preparation 2011: An Update©. Advances in Skin & Wound Care 24(9):415-36, quiz 437-8, 2011. Available at: http://www.ncbi.nlm.nih.gov/pubmed/21860264
119. Tickle, J. Wound Exudate Assessment and Management: A Challenge for Clinicians. British Journal of Nursing 24(Suppl 20):S38-43, 2015. Available at: http://www.magonlinelibrary.com/doi/10.12968/bjon.2015.24.Sup20.S38
120. Tamai, N., Akase, T., Minematsu, T., et al. Association Between Components of Exudates and Periwound Moisture-Associated Dermatitis in Breast Cancer Patients with Malignant Fungating Wounds. Biological Research for Nursing

18(2): 199-206, 2016. Available at: http://journals.sagepub.com/doi/10.1177/1099800415594452
121. Woo, K.Y., Beeckman, D., Chakravarthy, D. Management of Moisture-Associated Skin Damage: A Scoping Review. Advances in Skin & Wound Care 30(11):494-501, 2017.
122. Goto, T., Tamai, N., Nakagami, G., et al. Can Wound Exudate from Venous Leg Ulcers Measure Wound Pain Status? A Pilot Study. PLoS One 11(12):1-14, 2016.
123. Naylor, W.A. A Guide to Wound Management in Palliative Care. International Journal of Palliative Nursing 11(11):572-9, 2005. Available at: http://www.ncbi.nlm.nih.gov/pubmed/16471044
124. Benbow, M. Exploring Wound Management and Measuring Quality of Life. Journal of Community Nursing 22(6):14-8, 2008. Available at: https://www.jcn.co.uk/journal/06-2008/wound-management/1170-exploring-wound-management-and-measuring-quality-of-life/
125. Vowden, P., Bond, E., Meuleneire, F. Managing High Viscosity Exudate. Wounds International 6(1):14-8, 2015. Available at: www.woundsinternational.com
126. Woo, K., Santos, V.L.C.G., Alam, T. Optimising Quality of Life for People with Non-Healing Wounds. Wounds International 9(3):6-14, 2018. Available at: http://search.ebscohost.com/login.aspx?direct=true&db=rzh&AN=131918860&site=ehost-live
127. World Health Organization. Disability and Health 2018. Available at: https://www.who.int/en/news-room/fact-sheets/detail/disability-and-health
128. Faria, E., Blanes, L., Hochman, B., et al. Health-Related Quality of Life, Self-esteem, and Functional Status of Patients with Leg Ulcers. Wounds 23(1):4-10, 2011. Available at: https://www.woundsresearch.com/article/health-related-quality-life-self-esteem-and-functional-status-patients-leg-ulcers
129. McCaughan, D., Sheard, L., Cullum, N., et al. Patients' Perceptions and Experiences of Living with a Surgical Wound Healing by Secondary Intention: A Qualitative Study. International Journal of Nursing Studies 77:29-38, 2018. Available at: http://www.ncbi.nlm.nih.gov/pubmed/29031127
130. Moffatt, C.J., Franks, P.J., Doherty, D.C., et al. Psychological Factors in Leg Ulceration: A Case-Control Study. British Journal of Dermatology 161(4):750-6, 2009. Available at: http://www.ncbi.nlm.nih.gov/pubmed/19523173
131. Upton, D., Solowiej, K., Woo, K.Y. A Multinational Health Professional Perspective of the Prevalence of Mood Disorders in Patients with Acute and Chronic Wounds. International Wound Journal 11(6):627-35, 2014. Available at: http://doi.wiley.com/10.1111/iwj.12018
132. Upton, D., Solowiej, K., Hender, C., et al. Stress and Pain Associated with Dressing Change in Patients with Chronic Wounds. Journal of Wound Care 21(2):53-61, 2012. Available at: http://www.magonlinelibrary.com/doi/10.12968/jowc.2012.21.2.53

133. Nussbaum, S.R., Carter, M.J., Fife, C.E., et al. An Economic Evaluation of the Impact, Cost, and Medicare Policy Implications of Chronic Nonhealing Wounds. Value in Health 21(1):27-32, 2018. Available at: https://www.sciencedirect.com/science/article/pii/S1098301517303297
134. Ashford, R.I., McGee, P., Kinmond, K. Perception of Quality of Life by Patients with Diabetic Foot Ulcers. Diabetic Foot Journal 3:150-5, 2000.
135. Waters, N., Holloway, S. Diabetic Foot Disease on Employment. Diabetic Foot Canada 1(2):32-40, 2013.
136. Kapp, S., Miller, C., Santamaria, N. The Quality of Life of People Who Have Chronic Wounds and Who Self-Treat. Journal of Clinical Nursing 27 (1-2):182-92, 2018. Available at: http://doi.wiley.com/10.1111/jocn.13870
137. Ribu, L., Wahl, A. Living with Diabetic Foot Ulcers: A Life of Fear, Restrictions, and Pain. Ostomy/Wound Management 50(2):57-67, 2004. Available at: http://www.ncbi.nlm.nih.gov/pubmed/15129612
138. Edwards, H., Courtney, M., Finlayson, K., et al. A Randomised Controlled Trial of a Community Nursing Intervention: Improved Quality of Life and Healing for Clients with Chronic Leg Ulcers. Journal of Clinical Nursing 18(11):1541-9, 2009.
139. Fox, S. The Social Life of Health Information 2011. Available at: https://www.pewinternet.org/2011/05/12/the-social-life-of-health-information-2011/
140. Greene, J.A., Choudhry, N.K., Kilabuk, E., et al. Online Social Networking by Patients with Diabetes: A Qualitative Evaluation of Communication with Facebook. Journal of General Internal Medicine 26(3):287-92, 2011. Available at: http://www.ncbi.nlm.nih.gov/pubmed/20945113
141. Lorig, K., Ritter, P.L., Ory, M.G., et al. Effectiveness of a Generic Chronic Disease Self-Management Program for People with Type 2 Diabetes. The Diabetes Educator 39(5):655-63, 2013. Available at: http://journals.sagepub.com/doi/10.1177/0145721713492567
142. Nicholas, D.B., Fellner, K.D., Frank, M., et al. Evaluation of an Online Education and Support Intervention for Adolescents with Diabetes. Social Work in Health Care 51(9):815-27, 2012. Available at: http://www.ncbi.nlm.nih.gov/pubmed/23078013
143. van Rijswijk, L. The Language of Wounds. In: D. Krasner, G.T. Rodeheaver, R.G. Sibbald, eds. Chronic Wound Care: A Clinical Sourcebook for Health Care Professionals, 4th ed. Malvern, PA: HMP Communications, 2007:25-8.
144. Husband, L.L. Venous Ulceration: The Pattern of Pain and the Paradox. Clinical Effectiveness in Nursing 5(1):35-40, 2001. Available at: https://www.sciencedirect.com/science/article/pii/S1361900401901852
145. Chase, S.K., Melloni, M., Savage, A. A Forever Healing: The Lived Experience of Venous Ulcer Disease. Journal of Vascular Nursing 15(2):73-8, 1997. Available at: https://www.ncbi.nlm.nih.gov/pubmed/9238945

146. Corbett, L.Q., Ennis, W.J. What Do Patients Want? Patient Preference in Wound Care. Advances in Wound Care 3(8):537-43, 2014. Available at: http://www.ncbi.nlm.nih.gov/pubmed/25126474

2

Reimbursement Regulations Impacting Wound Care

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Objectives

After completing this chapter, you will be able to:

- discuss the significance of the U.S. Centers for Medicare and Medicaid Services
- discuss reimbursement issues related to hospitals, skilled nursing facilities, and home health agencies
- identify quality improvement efforts
- describe essential wound documentation required for reimbursement in the United States.

Chapter Overview

Reimbursement regulations in wound care as in any other sector of health care can be quite complex. This chapter is organized into five major sections, which are as follows: role of regulations in health care, government payers in wound care, principles of wound care reimbursement, wound care in different practice settings, and quality assessment and improvement issues.

Role of Regulation in Health Care

Regulations are a pervasive feature of the American healthcare system and, not surprisingly, significantly impact the delivery of wound care. Quite often, regulations and reimbursement determine who receives wound care and the level of wound care that is delivered. Thus, if clinicians are to provide optimum care, it is essential for them to have knowledge about the regulations that impact wound care in their specific practice setting.

Although many clinicians may view the current regulatory environment as burdensome and unnecessary, it's essential to recognize the important purpose that regulations fulfill. Healthcare regulations and standards are necessary to ensure compliance and to provide safe health care to every individual who accesses the system. The healthcare regulatory agencies in turn monitor practitioners and facilities, provide information about industry changes, promote safety, and ensure legal compliance and quality services.

Federal, state, and local regulatory agencies often establish rules and regulations for the healthcare industry, and their oversight is mandatory. Some other agencies, such as those for accreditation, require voluntary participation but are still important because they provide rankings or certification of quality and serve as additional oversight, ensuring that healthcare organizations promote and provide quality care.

In the case of wound care, the goal of current regulations is to ensure access to high-quality wound care, particularly for vulnerable populations such as the elderly and nursing home residents. As healthcare expenses continue to grow, wound care regulations also increasingly focus on limiting costs. Balancing the need to ensure quality while limiting costs can often be challenging.

Regulations are promoted at all levels of the government including federal, state, and local agencies. Additionally, nongovernment organizations such as accrediting bodies are responsible for the regulatory environment in which clinicians practice.

There are many types of regulatory vehicles available to the government to help achieve this goal. Government regulations may involve entry restrictions such as licensure and accreditations that seek to limit the ability to offer a particular service; they could use different payment methodologies that determine reimbursements for care provided; or they could involve quality controls that seek to improve the care that is provided.

There are many payers that reimburse for wound care such as those who are private, health plans (i.e., UnitedHealthcare, Aetna, Blue Cross/Blue Shield), unions, and employers, and those who are public (i.e., the Department of Veterans Affairs, Tricare). However, in this chapter, our focus is on the government payers in wound care. The major regulatory agency involved in wound care in the United States is the Centers for Medicare and Medicaid Services (CMS). In addition, we include an overview of how wound care is covered, coded, and reimbursed by CMS and its contractors; the different reimbursement scenarios depending on the clinicians' practice settings; and a description of the agency's efforts in improving the quality of wound care. Through these efforts, CMS aims to improve health and health care while also making care more affordable.

General Information on Government Payers in Wound Care

Centers for Medicare and Medicaid Services

CMS is a federal agency within the U.S. Department of Health and Human Services. Prior to July 1, 2001, it was called the Health Care Financing Administration (HCFA). CMS administers the Medicare and Medicaid programs. Moreover, because CMS provides the states with at least 50% of their finances for healthcare costs, the states must comply with federal regulations.

The difference is that while both the Medicare and Medicaid programs are administered through federal statutes that determine beneficiary requirements, what is covered, payment fees and schedules, and survey processes of clinical settings (such as skilled nursing facilities [SNFs] or home health agencies [HHAs]) are determined by their respective programs. Both programs have a wide variance on coverage, eligibility, and payment fees and schedules. Therefore, it's important for the clinician to know what's covered and the level of reimbursement prior to developing a treatment plan with the patient. Because CMS remains the largest health insurance agency, many private insurance companies will provide coverage at similar levels.

Medicare

The Medicare program was developed in 1965 by the federal government.[1] In order to qualify for Medicare benefits, a person must be age 65 or older, have approved disabilities if under age 65, or have end-stage renal disease.

In 2017, Medicare provided coverage to more than 55.7 million people[2], spending $705.9 billion on benefits.[3] These benefit payments are funded from two trust funds—the Hospital Insurance (HI) Trust Fund and the Supplementary Medical Insurance (SMI) Trust Fund. Most often, these are referred to as Medicare Part A and Medicare Part B, respectively.[4]

The HI Trust Fund pays for a portion of the costs of inpatient hospital services and related care. Those services include critical access hospitals (small facilities that give limited outpatient and inpatient services to people in rural areas), SNFs, hospice care, and some home healthcare services. The HI Trust Fund is financed primarily through payroll taxes, plus a relatively small amount of interest, income taxes on Social Security benefits, and other revenues.

The SMI Trust Fund pays for a portion of the costs of physicians' services, outpatient hospital services, and other related medical and health services. As of 2019, the premium for Medicare Part B is $135.50 per month.[5] In some cases, this amount may be higher if the person doesn't choose Medicare Part B when he or she first becomes eligible at age 65 or if the person files taxes greater than $85,000 as an individual or $170,000 as part of a couple. In addition, as of January 2006, the SMI Trust Fund pays for private prescription drug insurance plans to provide drug coverage under Part D of the program. The separate Part B and Part D accounts in the SMI Trust Fund are financed through general revenues, beneficiary premiums, interest income, and, in the case of Part D, special payments from the states.

The Medicare+Choice program was authorized by the Balanced Budget Act of 1997.[6] In this program, beneficiaries have the traditional Medicare Part A and Part B benefits, but they may also select Medicare managed care plans (such as health maintenance organizations [HMOs], preferred provider organizations [PPOs], or private fee-for-service [FFS] plans). Medicare+Choice plans provide care under contract to Medicare. They may provide benefits such as coordination of care or reducing out-of-pocket expenses. Some plans may also offer additional benefits, such as prescription drugs.

Prescription drug benefits are available for all Medicare beneficiaries regardless of income, health status, or prescription drug use[6] through Medicare Part D. A range of plans are available, so beneficiaries have multiple options for coverage. Moreover, persons can add drug coverage to the traditional Medicare plan through a "stand-alone" prescription drug plan or through a Medicare Advantage

plan, which includes an HMO or PPO and typically provides more benefits at a significantly lower cost through a network of doctors and hospitals. Presently, no wound care products are covered under this benefit.

Medicaid

The Medicaid program was developed in 1965 as a jointly funded cooperative venture between the federal and state governments to assist states in the provision of adequate medical care to eligible people.[1] Medicaid is the largest program providing medical and health-related services to America's poorest people. Within broad national guidelines provided by the federal government, each of the states:

- administers its own program
- determines the type, amount, duration, and scope of services
- establishes its own eligibility standards
- sets the rate of payment for services
- determines what products are covered in that state.

Thus, the Medicaid program varies considerably from state to state as well as within each state over time. This wide variance also affects what is covered in wound care. For example, the number of times debridement of a wound is reimbursed differs by state, as do product treatment options.

Managed Care Organizations

Managed care organizations (MCOs) were developed to provide health services while controlling costs. They combine the responsibility for paying for a defined set of health services with an active program to control the costs associated with providing those services, while at the same time attempting to control the quality of and access to those services. The health benefits, which usually range from acute care services to dental and vision coverage, are usually clearly identified, as are the payment, copayment, and deductibles that are required for a specific health procedure (e.g., compression therapy for chronic venous insufficiency ulcer).

Moreover, the MCO usually receives a fixed sum of money to pay for the benefits in the plans for the defined population of enrollees. Typically, this fixed sum is constructed through premiums paid by the enrollees, capitation payments made on behalf of the enrollees from a third party, or both. There are wide variations in MCOs and the services they provide for patients with wounds.

Economic Impact of Chronic Wounds in the Medicare Program

Chronic nonhealing wounds are a major contributor to the Medicare budget. A study, *An Economic Evaluation of the Impact, Cost, and Medicare Policy Implications of Chronic Nonhealing Wounds*,[7] published in the 2018 *Value in Health* journal demonstrates this impact in Medicare patients. The study analyzed the Medicare 5% Limited Data Set for calendar year 2014 and determined that chronic nonhealing wounds impact nearly 15% (8.2 million) of Medicare beneficiaries, far more than suggested by previous studies. Furthermore, conservative estimates for total Medicare annual spending for all wound types ranged from $28.1 billion to $31.7 billion. Treatment and management of infected or reopened (dehisced) surgical wounds account for the highest per wound costs. Hospital outpatient care drove the highest site-of-service costs, demonstrating the shift from hospital inpatient to outpatient services in the wound care space.

Practice Point

Infected or dehisced surgical wounds treatment account for the highest per wound costs in the United States.

The findings highlight the need for federal research funding, quality measures, and reimbursement models that are relevant to wound care. Such measures are not currently included under the Centers for Medicare and Medicaid Services (CMS) payment policies, including the Medicare Access and CHIP Reauthorization Act (MACRA).

In addition, the documentation of the specific, significant burden of chronic wounds in the Medicare population illustrates the need for CMS and health policy makers to include wound-relevant quality measures in all care settings as well as develop episode of care measures, chronic care models, and reimbursement models to drive better health outcomes and smarter spending in the wound care space.

General Wound Care Reimbursement Principles

Reimbursement directly impacts how clinicians deliver care. Increasingly, payers (Medicare, Medicaid, HMOs) are examining where their money is going and whether they are getting the most from providers on behalf of their beneficiaries. Thus, payers are requiring more documentation regarding patient outcomes to justify payment. Clinicians who can document comprehensive and accurate assessments of wounds and the outcomes of their interventions are in a stronger position to obtain and maintain coverage and thus reimbursement.

Evidence-based wound care should always be the goal of clinicians. However, clinicians are increasingly being challenged to provide optimum wound care based on healthcare setting and payers.

Medicare reimbursement is more than just the payment for medical items and services. The key to understanding how Medicare reimburses providers, physicians, and suppliers for wound care involves a greater understanding of three main components that comprise the Medicare reimbursement system: coding, coverage, and payment. Each is a separate and distinct process. Just because a product is awarded a code does not mean it will be covered. Just because it is awarded a code and covered does not mean it will be reimbursed. Similarly, all procedures performed by clinicians have codes assigned to them and are reimbursed based on the payment system for the setting in which it was performed.

Since coding and coverage are universal to all settings, they will be discussed first. Then, we will discuss reimbursement in a setting-specific fashion.

Coding

In order for medical claims to be processed, billing codes are used by physicians, hospitals, and other providers to identify the diagnosis, product, service, and procedure that the clinician used in treating the patient in which they are billing a payer. Accurate coding is necessary in order for the claim to be properly and accurately processed.

The types of codes that are used include:

- Healthcare Common Procedure Coding System (HCPCS) Level I and Level II
- Diagnosis-related group (DRG)
- International Classification of Diseases (ICD-10)

HCPCS Level I

HCPCS Level I or Current Procedural Terminology (CPT®) codes are numbers assigned to a procedure that a clinician (e.g., physician, nurse practitioner, podiatrist) may perform on a patient, including medical, surgical, and diagnostic services. The codes are then used by insurers (Medicare, Medicaid, and private payers) to determine the amount of reimbursement for the clinician. Every clinician uses the same codes to ensure uniformity, but the amount of reimbursement may differ depending on their profession. An example of a CPT code for wound care is CPT 11042—debridement, subcutaneous tissue (includes the epidermis and dermis, if performed; first 20 sq. cm or less).

HCPCS Level II

HCPCS Level II code set is made up of five-character alphanumeric codes representing primarily medical supplies, durable medical goods, nonphysician services, and services not represented in the Level I code set (CPT). HCPCS Level II includes services and products such as ambulance, durable medical equipment (DME), prosthetics, orthotics, and supplies (DMEPOS) when used outside a physician's office. *Cellular and/or Tissue-Based Products for Skin Wounds* (CTPs), an updated and more clinically appropriate term for

“skin substitutes,” surgical dressings, support surfaces, and negative pressure wound therapy (NPWT), all have HCPCS Level II codes.

Diagnosis-Related Group

DRGs are used for inpatient hospital claims. DRGs are a means of classifying a patient under a particular group where those assigned are likely to need a similar level of hospital resources for their care. This allows hospital administrators to more accurately determine the type of resources needed to treat a particular group and to predict more closely the cost of that treatment.

International Classification of Diseases

ICD-10 is a set of codes used by physicians, hospitals, and allied health workers to indicate diagnosis for all patient encounters. The ICD-10-CM coding system contains over 68,000 codes and is much more specific than the ICD-9. For example, ICD-10-CM codes for pressure ulcers/injuries describe location, stage, and whether present on admission (POA).

Coverage

Coverage is the existence of a medical benefit category for a service, procedure, device, drug, or supply used in healthcare delivery. Coverage varies by the type of health plan (i.e., Medicare, Medicaid, private payers, etc.), the setting of care (i.e., hospital, home health, SNF, physician office, wound care clinic, etc.), and the condition of the patient. If coverage is permissible, payers may have a separate coverage policy that will dictate the specific criteria in which they will permit coverage of that product, service, or procedure. The coverage policy will set forth medical conditions, diagnosis, coding, and specific requirements and/or limitations for coverage of that particular service or product. This will dictate whether it will get paid.

The different settings by which coverage may be permitted for wound care includes hospitals (inpatient, outpatient, and long-term care hospitals) and outpatient clinics—including wound care clinics, SNFs, and physician offices and also home care.

In the Medicare program, there are few national coverage policies (NCD) for the products that would be used to treat a patient with a chronic wound. Rather, coverage for most wound care products and procedures are made through local coverage determinations (LCDs) and policy articles by the Medicare Administrative Contractors (MACs). The Part A and B Medicare Administrative Contractors (A/B MACs) create coverage policies for CTPs, disposable NPWT, and debridement. The Durable Medical Equipment Medicare Administrative Contractors (DMEMACs) create coverage policies for DME prosthetics, orthotics, and supplies (DMEPOS). The policies they create in the wound care space include surgical dressings, NPWT, pneumatic compression devices, and support surfaces. Wound care–specific national coverage decisions issued by CMS include hyperbaric oxygen treatments, topical oxygen, and general coverage information for pneumatic compression devices. Both the national and local coverage policies and policy articles can be found on both the CMS and specific contractor's website: https://www.cms.gov/medicare-coverage-database/overview-and-quick-search.aspx.[8]

The following is additional information regarding the CMS contractors who create and implement the LCDs—the DMEMACs and the A/B MACs

Medicare Administrative Contractors

MACs are private healthcare insurers that have been awarded a geographic jurisdiction to process Medicare Part A and Part B (A/B) medical claims or DME claims for Medicare FFS beneficiaries. CMS relies on a network of MACs to serve as the primary operational contact between the Medicare FFS program and the healthcare providers enrolled in the program. MACs are multistate, regional contractors responsible for administering both Medicare Part A and Medicare Part B claims. MACs perform many activities including:

- Process Medicare FFS claims
- Make and account for Medicare FFS payments
- Enroll providers in the Medicare FFS program
- Handle provider reimbursement services and audit institutional provider cost reports
- Handle redetermination requests (first stage appeals process)
- Respond to provider inquiries
- Educate providers about Medicare FFS billing requirements
- Establish LCDs
- Review medical records for selected claims
- Coordinate with CMS and other FFS contractors

The MACs serve more than 1.5 million healthcare providers enrolled in the Medicare FFS program. Collectively, the MACs process more than 1.2 billion Medicare FFS claims annually, 218 million Part A claims, and more than 1 billion Part B claims and paid $386 billion in Medicare benefits.

Durable Medical Equipment Medicare Administrative Contractors

The DMEMACs are responsible for handling the administration of Medicare claims from DMEPOS suppliers. The DMEMACs serve as the point of contact for all Medicare suppliers, whereas beneficiaries

can register their claims-related questions to Beneficiary Contact Centers. Currently, there are four DMEMACs (Table 2-1).

Table 2-1 The Four Durable Medical Equipment Medicare Administrative Contractors

- *JA—Noridian Healthcare Solutions LLC*, serving Connecticut, Delaware, the District of Columbia, Maine, Maryland, Massachusetts, New Hampshire, New Jersey, New York, Pennsylvania, Rhode Island, and Vermont
- JB—Cigna Government Services (CGS) Administrators, serving Illinois, Indiana, Kentucky, Michigan, Minnesota, Ohio, and Wisconsin
- JC—Cigna Government Services (CGS), serving Alabama, Arkansas, Colorado, Florida, Georgia, Louisiana, Mississippi, New Mexico, North Carolina, Oklahoma, Puerto Rico, South Carolina, Tennessee, Texas, U.S. Virgin Islands, Virginia, and West Virginia
- *JD—Noridian Healthcare Solutions LLC* serving Alaska, American Samoa, Arizona, California, Guam, Hawaii, Idaho, Iowa, Kansas, Missouri, Montana, Nebraska, Nevada, North Dakota, Northern Mariana Islands, Oregon, South Dakota, Utah, Washington, and Wyoming

DMEMACs clearly define local medical coverage policies. The beneficiary usually pays a deductible for Medicare Part B, which in 2019 is the first $185.00 for covered medical services annually. Once

that has been met, the beneficiary pays 20% of the Medicare-approved amount for services or supplies. If services weren't provided on assignment, then the beneficiary pays for more of the Medicare coinsurance plus certain charges above the Medicare-approved amount.

Examples of Coverage Policies for Certain Wound Care Products

Here are examples of coverage policies for certain wound care products:

- Negative pressure wound therapy—Medicare Part B provides coverage for NPWT pumps. In order for a NPWT pump and supplies to be covered, the patient must have a chronic stage 3 or 4 pressure injury, neuropathic ulcer, venous or arterial insufficiency ulcer, or a chronic (at least 30 days) ulcer of mixed etiology. Extensive documentation is required prior to a DMEMAC approving coverage for NPWT. Thus, it is important for the clinician to review the coverage policy and policy article to ensure that the product is covered under the Medicare program.[8]
- Support surfaces are also covered under Medicare Part B.[8] CMS has divided support surfaces into three categories for reimbursement purposes:
    - Group 1 devices include pads or mattresses that are comprised of air, gel, water, foam, or a powered pressure-reducing alternating mattress overlay/pad.
    - Group 2 devices include powered pressure-reducing mattress or overlay (alternating pressure, low air loss, or powered flotation without low air loss) and advanced nonpowered pressure-reducing mattress or overlay.
    - Group 3 devices comprise only air-fluidized beds.

Specific criteria must be met before Medicare will reimburse for support surfaces; therefore, it is essential for the clinician to review both the LCD and policy article.[8]

- Surgical dressings—The surgical dressing benefit covers primary and secondary dressings in outpatient care clinic settings (e.g., a hospital outpatient wound center) and physician offices (Table 2-2). This coverage policy is determined by the DMEMACs as well.
- Pneumatic compression devices are covered when treating a patient with chronic venous insufficiency with venous stasis ulcers or lymphedema. Reimbursement for these items is based upon the coverage criteria based in the LCD. The policy is very detailed and needs to be reviewed for specific coverage language when providing these devices to patients.

Table 2-2 Coverage Under the Surgical Dressings Benefit

To have the dressings reimbursed under the Medicare/Medicaid surgical dressings benefit, the following criteria must be met:

- The dressings are medically necessary for the treatment of a wound caused by, or treated by, a surgical procedure.
- The dressings are medically necessary when debridement of a wound is medically necessary.

In certain situations, dressings aren't covered under the surgical dressings benefit, including those for:

- drainage from a cutaneous fistula that has not been caused by or treated by a surgical procedure
- first-degree burn
- stage 1 pressure injury
- wounds caused by trauma that don't require surgical closure or debridement (such as skin tears and abrasions)
- venipuncture or arterial puncture site other than the site of an indwelling catheter or needle.

Examples of dressing classifications that are covered under the surgical dressing benefit include but not limited to:

- foam dressings
- gauze
- nonimpregnated and impregnated dressings
- hydrocolloids
- alginates
- composites
- hydrogels
- collagen
- compression bandages and multilayer systems.

A/B Medicare Administrative Contractors

A/B MACs carry out the administrative responsibilities of traditional Medicare for Part A and B. They are responsible for claims processing or cutting checks to Medicare providers for their services; ensuring services are correctly coded and billed for, both before and after payment; deciding which healthcare services are medically necessary; and collecting overpayments. MACs follow the national coverage determinations set by the CMS, but in cases where there is no such determination or the rules are too vague regarding a specific procedure, a MAC may develop a LCD. The coverage policies for cellular and/or tissue products for skin wounds (CTPs) are administered through these A/B MAC contractors in their local jurisdictions within LCDs. In these policies, the coverage parameters can vary from one jurisdiction to another as can the title of the policy itself. The clinician would need to refer to each of the jurisdictions in order to understand whether CTPs are covered in their jurisdiction and their parameters for coverage. In addition to CTPs, LCDs related to disposable NPWT and debridement are also provided by the MACs in their wound care policies. Coverage policies for these areas are not consistent from one jurisdiction to another either and as such clinicians should review the coverage policies for their particular jurisdiction. There are currently 12 jurisdictions (Table 2-3).

Table 2-3 The 12 AB Medicare Administrative Contractors

Jurisdiction E—Noridian Healthcare Solutions, serving American Samoa, California, Guam, Hawaii, Nevada, and the Northern Mariana Islands

Jurisdiction F—Noridian Healthcare Solutions serving Alaska, Arizona Idaho, Montana, North Dakota, Oregon, South Dakota, Utah, Washington, and Wyoming

Jurisdiction H—Novitas Solutions serving Arkansas, Colorado, Louisiana, Mississippi, New Mexico, Oklahoma, and Texas

Jurisdiction J—Palmetto Government Benefits Administrators serving Alabama, Georgia, and Tennessee

Jurisdiction K—National Government Services serving Connecticut, Maine, Massachusetts, New Hampshire, New York, Rhode Island, and Vermont

Jurisdiction L—Novitas Solutions serving Delaware, the District of Columbia, Maryland, New Jersey, and Pennsylvania

Jurisdiction M—Palmetto Government Benefits Administrators serving North Carolina, South Carolina, Virginia, and West Virginia

Jurisdiction N—First Coast Service Options serving Florida, Puerto Rico, the U.S. Virgin Islands

Jurisdiction 5—Wisconsin Physicians Service Iowa, Kansas, Missouri, and Nebraska

Jurisdiction 6—National Government Services serving Illinois, Minnesota, Wisconsin

Jurisdiction 8—Wisconsin Physicians Service serving Indiana and Michigan

Jurisdiction 15—Cigna Government Services Kentucky, Ohio

Payment

Payment refers to the methodology used to determine reimbursement to a healthcare provider or supplier. Payment may take the form of a global or bundled payment for the combination of services needed to treat a particular condition, as is the case with many hospital inpatient and outpatient discharges, or may be made on an itemized basis, as is the case for many physician services such as office visits. In many cases, the payment method will be determined by the site of service rather than by the item or service itself.

As mentioned, since wound care is provided in multiple settings, a later section on “How Reimbursement Works in Clinicians’ Practice Settings” is devoted to the various healthcare settings and how wound care products and services are reimbursed by CMS.

Important Shift in Clinician Payment

Historically, providers and healthcare organizations have been compensated for care provided based on FFS reimbursement models. When a provider is reimbursed based on a FFS model, they are compensated for each procedure, test, treatment, etc. they perform, regardless of whether that procedure, test, or treatment results in a better outcome for the patient.

Essentially, with the FFS model, providers are financially rewarded for quantity over quality. With this payment model, it's easy to see how patients can sometimes undergo unnecessary tests or treatments when perhaps less invasive, lower-cost, and just-as-effective options are available.

When congress enacted the Medicare Access and CHIP Reauthorization Act (MACRA) in 2015, it changed the way physicians got paid by repealing the unpopular sustainable growth rate (SGR) formula and created a shift from a fee-for-service reimbursement model to one that is value-based.

Medicare will allow physicians to choose between two payment tracks starting in 2019. The first track, the Merit-Based Incentive Payment System (MIPS), more closely resembles previous Medicare payment methods. Physicians are reimbursed primarily via FFS, with relative payment rates for each service determined by the resource-based relative value scale. Physicians will also receive bonuses or penalties related to their performance. Performance-based payments will be based on quality of care, resource use, meaningful use of electronic health records (EHRs), and clinical practice improvement, replacing several previous Medicare physician incentive programs.

The second payment track includes physicians with significant participation in certain alternative payment models (APMs). In contrast to FFS, value-based APMs compensate providers not for the quantity of procedures performed but rather for the quality of the care they provide, measured by patient health outcomes as well as quality care, improved health, and lower costs. In a value-based

reimbursement model, providers are rewarded for effectively managing the health of individuals and populations.[9]

In 2017, 34% of total U.S. healthcare payments were tied to these APMs, which have these categories:[10]

- 41% of healthcare dollars in Category 1 (FFS—No Link to Quality & Value)
- 25% of healthcare dollars in Category 2 (FFS—Link to Quality & Value)
- 34% of healthcare dollars in Categories 3 (APMs Built on FFS Architecture) and 4 (Population-Based Payment)

Value-based care requires providers to take a more team-oriented approach to patient care, coordinating care across the continuum and collaborating with a patient's other care providers to deliver the best health outcomes possible.

Correct Documentation Is Key for Payment

Comprehensive documentation (see Table 2-4) is the critical foundation for successful reimbursement of services and products. Physician documentation of debridement as well as the application of CTPs is important to ensure coverage and payment. Moreover, physician documentation of pressure ulcers/injuries at the time of hospitalization is particularly important for identifying POA status. Regulatory agencies, independent of their healthcare settings, set forth the requisite documentation for reimbursement, and their requirements for documentation should always be carefully reviewed prior to applying for coverage. Thorough documentation justifies the medical necessity of services and products and should reflect the care required in the prevention or treatment of wounds. Without adequate documentation, providers run the risk that the product or service being provided will be denied or reimbursement withheld. In order to promote better documentation for pressure ulcers/injuries as well as the availability of data necessary for quality measurement, CMS is also promoting the use of EHRs.[11] Specific financial incentives are available to providers using EHRs and reporting data on quality of care.

Table 2-4 Essential Wound Documentation

For essential wound care documentation, include the following:

- Change in clinical status or wound healing progress
- Characteristics of the wound, including:
  - Location
  - Length, width, and depth
  - Staging/category/classification
  - Exudate amount
  - Tissue type
  - Pain
- Local wound care and dressing selection
- Nutritional status
- Pressure redistribution/support surfaces (both bed and chair)
- Outcome and Assessment Information Set (OASIS-C2) per schedule in home health care
- Minimum Data Set (MDS 3.0) per schedule in SNF
- Regular assessment and reassessment of the wound (such as daily or weekly)
- Repositioning schedule
- Routine daily skin assessment and care

For other areas in wound care that are governed by the A/B MACs such as debridement and CTPs, it is imperative to be aware of and review the coverage policies that impact your practice as they do set forward documentation requirements that need to be strictly adhered to. Finally, as of July 24, 2017, the DMEMACs issued a specific LCD, which specifically addresses documentation for all DMEPOS claims submitted to them—for wound care, this would include surgical

dressings, NPWT, pneumatic compression, etc. The documentation requirements must be followed in order to gain coverage and ultimately reimbursement for those products provide to your patients.[12]

How Reimbursement Works in Clinicians' Practice Settings

Hospital Inpatient

Hospital reimbursement is part of the inpatient prospective payment system (IPPS). Payments made under the IPPS totaled $112 billion in 2015.[13] The inpatient benefit covers beneficiaries for 90 days of care per episode of illness. There is a 60-day lifetime reserve. The episode of care begins when the Medicare beneficiary is admitted to the hospital and ends when he or she has been out of the hospital or an SNF for 60 consecutive days.

Under the IPPS, each hospital discharge is assigned to 1 of 335 different DRGs based on the ICD-10 diagnostic code and procedure codes. Each DRG may be further divided into two or three Medicare Severity DRGs (MS-DRG) based on the presence of specific comorbidities or complications. Hospitals are reimbursed on a predetermined, lump-sum fixed rate for each MS-DRG. For hospitals, this payment would also include all medical care, procedures, surgeries, wound care products, devices, and support surfaces. Because the IPPS is based on an adjusted average payment rate, some cases will receive payments in excess of cost (less than the billed charges), whereas others will receive payment that's less than cost. The system is designed to give hospitals the incentive to manage operations more efficiently by evaluating those areas in which increased efficiencies can be instituted without affecting the quality of care and by treating a mix of patients to balance cost and payments. CMS does revise annually the rules for its IPPS.

Increasingly, CMS is emphasizing value-based purchasing, and there are at least three different programs that will modify the amount that a hospital receives for any given case.[13] Under the Hospital Readmissions Reduction Program, hospitals with risk-adjusted readmission rates above the national average will have their IPPS reduced by as much as 3%. The Hospital Value-Based Purchasing Program looks at a broad number of quality, safety, and satisfaction measures in awarding an incentive to high-performing hospitals.[14] Under the Hospital-Acquired Condition (HAC) Reduction Program, 25% of hospitals with the highest rates of avoidable complications,

such as falls and catheter-associated urinary tract infections, receive a 1% reduction in payments.

Wounds are considered in these different quality programs. Both stage 3 and 4 pressure ulcers/injuries and surgical site infections are considered as HACs that are used in both the Value-Based Purchasing Program and the HAC Reduction Program. Rates are calculated from the Patient Safety Indicators (PSIs) software, which uses a highly selective algorithm to determine which wounds are counted.[15,16] The pressure injury rates in the PSIs consider only stage 3 and 4 pressure injuries and excludes wounds occurring in certain high-risk populations such as nursing home residents and people with spinal cord injuries. The impact of these reimbursement programs on pressure ulcer/injury rates remains uncertain.[16] In 2008, as part of the HACs Initiative, CMS eliminated comorbidity payments to hospitals for stage 1 and 2 pressure injuries that were POA. The financial impact on hospitals of this change was 200 times greater than the impact of removing payments for hospital-acquired stage 3 or 4 pressure injuries.[17] These different programs emphasize the importance of clinicians identifying and subsequently documenting the pressure injuries or specific surgical site infection that are POA.

Hospital Outpatient Centers

The Balanced Budget Act of 1997 provided authority for CMS to develop a prospective payment system (PPS) under Medicare for hospital outpatient services. The new outpatient PPS took effect in August 2000.[18] All services paid under this PPS are placed into ambulatory payment classifications (APCs). A payment rate is established for each APC, depending on the services/procedures provided. CPT codes and modifiers identify clinic visits and services/procedures. The CPT codes track to an APC group based on the cost and the level of resources required to perform the service or procedure. Services or procedures in each APC are similar in cost. Since hospital outpatient claims are submitted to the Part B MAC, the MAC pays a predetermined amount for the APC group—which includes all supplies including, but not limited to, wound care dressings. Beginning in 2013, CMS determined that cellular- and tissue-based products for skin wounds (CTPs) should be bundled. As a result, these products are now packaged into the facility fee for the procedure. At time of publication, CMS is considering other payment methodologies for CTPs. They are seriously considering an episode of care method of payment. Whatever methodology CMS decides to adopt, they are targeting 2020 for implementation.

Hospitals may be paid for more than one APC per encounter. Medicare beneficiaries also can pay a coinsurance, which is the amount they will have to pay for services furnished in the hospital outpatient department after they have met the Medicare Part B deductible. A coinsurance amount is initially calculated for each APC based on 20% of the national median charge for services in the APCs. The coinsurance amount for an APC doesn't change until the amount becomes 20% of the total APC payment. It should be noted that the total APC payment and the portion paid as coinsurance amounts are adjusted to reflect geographic wage variations using the hospital wage index and assuming that the portion of the payment/coinsurance that's attributable to labor is 60%.

Skilled Nursing Facilities

A patient who is eligible for Medicare may receive Medicare Part A for up to 100 days per benefit period in an SNF.[19] The patient must satisfy specific rules in order to qualify for this benefit. These rules include the following:

- Beneficiary is admitted to SNF within 30 days after the date of hospital discharge.
- Beneficiary must have been in a hospital receiving inpatient hospital services for at least 3 consecutive days.
- A physician determines that the beneficiary requires skilled nursing care by or under the supervision of a registered nurse or requires physical, occupational, or speech therapy that can only be provided in an inpatient setting.
- Services are needed on a daily basis.
- Skilled services are required for the same or related health problem that resulted in the hospitalization.

Since 1998, SNFs have been paid on the basis of a PPS. The PPS payment rates are adjusted for case mix and geographic variation (urban vs. rural) in wages. The PPS also covers all costs of furnishing covered SNF services. Thus, wound care supplies, therapies, and support surfaces are included in the PPS per diem rate, which could limit the incentive to provide more expensive wound care therapies.[20] The SNF isn't permitted to bill under Medicare Part B until the 100 days are in effect.

Long-term care is among the most heavily regulated industries. All SNFs participating in Medicare and Medicaid must comply with these federal and state regulations, commonly referred to as federal (or F)-Tags.[21] In November 2017, CMS released its revised interpretative guidelines to clarify requirements and assist with the survey process. Requirements for chronic wounds emphasize the proper identification of the type of wound and development and implementation of a comprehensive plan of care. Requirements for pressure ulcers/injuries are particularly extensive and are specified in F-Tag 686 (before 2017 it was F-Tag 314). This emphasizes that:

- A resident receives care, consistent with professional standards of practice, to prevent pressure ulcers/injuries and does not develop pressure ulcers/injuries unless the individual's clinical condition demonstrates that they were unavoidable.
- A resident with pressure ulcers/injuries receives necessary treatment and services, consistent with professional standards of practice, to promote healing, prevent infection, and prevent new ulcers from developing.

SNFs that are found to be noncompliant with these regulations can receive civil money penalties of several thousand dollars per day, or CMS and the state can withhold payments and close the facility because of system-wide imminent danger to residents.

Resident Assessment Instrument

In order to improve care, CMS requires that a standardized assessment consisting of the resident assessment instrument (RAI) be completed on all SNF residents. Central to the assessment process is the Minimum Data Set (MDS), a 400-item form that attempts to identify the functional capacity of residents in SNFs. Among the different dimensions captured during the assessment are functional status, cognition, continence, active medical conditions, and skin condition. Based on these assessments, further evaluations are triggered and care plans developed. The MDS is completed on admission, quarterly, and with significant changes in health status.[22]

The current MDS version 3.0 has been in use since 2010. This revised version was intended to improve reliability, accuracy, and usefulness when compared to earlier versions. Significant changes were made, in particular, to Section M on skin conditions.[23,24] Two subsections describe number of venous and arterial ulcers as well as the presence of other skin conditions such as burns, skin tears, surgical wounds, and moisture associated skin damage. The remaining nine subsections are dedicated to pressure ulcers/injuries. Information is collected on risk of pressure ulcers/injuries, the number of ulcers at various stages, the most severe tissue type, and whether there is worsening of pressure injury status. With MDS 3.0, CMS also adapted the National Pressure Injury Advisory Panel's staging categories with the inclusion of deep tissue injuries and unstageable pressure injuries. Healing of pressure injuries is better described so that there is no need for reverse staging. Since implementation of MDS 3.0, there have been ongoing updates. Specific directions in 2016 helped clarify under what conditions a pressure injury is classified as POA to the nursing home.[24] Staging of blistering pressure injuries has also undergone revisions.[25]

Based on the responses in Section M, specific actions may be triggered that address pressure injuries, nutritional status, or dehydration/fluid maintenance. The net result of these changes is closer linkage of the resident assessment to quality of life,

incorporation of updated guidelines for ulcer staging, and broadening of the care planning process to include current clinical protocols and evidence-based standards.

Resource Utilization Groups

The RAI is also linked to Medicare SNF payment using a case-mix classification system. This PPS creates a per diem rate that covers most SNF services including wound care. Based on the MDS, each resident is assigned to 1 of 66 Resource Utilization Groups (RUGs).[26,27] RUGs is a classification system that assigns nursing home residents to different groups depending on resident characteristics and specific therapies being received. Each RUG category is considered to be relatively homogeneous with regard to expected resource utilization. The classification system includes 14 rehabilitation groups, 9 groups for days with rehabilitation and extensive services, 3 groups for extensive services, 16 groups for special care, and 10 groups for clinically complex care. Wound care is typically within the special care group. RUG rates are computed separately for urban and rural areas, and a portion of the total rate is adjusted to reflect labor market conditions in each SNF's location. Because of RUGs, it is essential for the SNF to complete the MDS correctly. The SNF must pay close attention to all health problems of the resident because the more intensive the care required, the higher the daily rate will be. Moreover, completing the MDS accurately and in a timely manner will help to ensure correct payments. If a SNF doesn't complete the MDS in a timely manner, it receives a default payment, which is usually significantly lower, or it may not receive payment at all.

Beginning in late 2019, CMS is updating its PPS for SNFs. There is an increased emphasis on value-based purchasing so that facilities receive a positive or negative financial incentive based on how well they prevent hospital readmissions.[28] Additionally, rather than relying on RUGs, there is a new case-mix–based PPS called the Patient-Driven Payment Model, which aims to base payments more on resident clinical characteristics than on specific services being received.

Home Health Agencies

Home health services, including skilled nursing and home health aide services, physical therapy, occupational therapy, and speech therapy, are also covered by Medicare. The Balanced Budget Act of 1997 called for the development of a PPS for Medicare home health services, which was implemented on October 1, 2000.[29] Beneficiaries receiving home health care are typically restricted to their homes, need skilled care on a part-time or intermittent basis, and are not required to make any copayments for these services.

Medicare currently purchases home health services in units of 60 days—or episodes of care. To capture the differences in expected resource use, patients are assigned to 1 of 153 home health resource groups (HHRGs) based on their clinical and functional status and service use as measured by the Outcome and Assessment Information Set (OASIS). There is a base rate for the HHRGs, which is then adjusted based on geographic factors. Reimbursements for routine and nonroutine medical supplies are included in this base rate for every Medicare home health patient. However, the HHRG payment does not include drugs, biologics, or DME—including but not limited to NPWT and pressure-relieving devices. Since 2017, a separate payment is made to HHAs for NPWT using a disposable device for a patient under the home health benefit. This was included in the Consolidated Appropriations Act, 2016 (Pub. L 114-113), which requires a separate payment to be made to HHAs for disposable NPWT devices when furnished, on or after January 1, 2017, to an individual who receives home health services for which payment is made under the Medicare home health benefit.

Disposable NPWT services are billed using the following CPT® codes:

- 97607—NPWT (e.g., vacuum-assisted drainage collection), utilizing disposable, nonDME including provision of exudate management collection system, topical application(s), wound assessment, and instructions for ongoing care, per session; total wound(s) surface area less than or equal to 50 sq. cm.

- 97608—NPWT (e.g., vacuum-assisted drainage collection), utilizing disposable, nonDME including provision of exudate management collection system, topical application(s), wound assessment, and instructions for ongoing care, per session; total wound(s) surface area greater than 50 sq. cm.

These codes include both performing the service and the disposable NPWT device. Payment for disposable would be made under the Hospital Outpatient Prospective Payment System (HOPPS); therefore, the payment amount will also be subject to the area wage adjustment policies in place under the OPPS in a given year.

CMS created an educational article called MedLearn (MLN) Matters to inform home health agencies how to bill for these products.[30]

Furthermore, in July 2018, CMS proposed a revision to the PPS for Medicare home health care that is intended to be implemented in 2020.[31] The Patient-Driven Groups Model would remove the current incentive to overprovide therapy and, instead, is designed to rely more heavily on clinical characteristics and other patient information to allow payments to more closely coincide with patients' needs. The unit of payment would also be changed to a 30-day episode of care.

OASIS-C2

When it is determined that a Medicare patient can receive home health services, an OASIS form must be completed. OASIS is a group of comprehensive assessments that are the basis for delivering patient care, systematically measuring patient outcomes with appropriate adjustment for risk factors affecting those outcomes, and, since 2000, assisting in the PPS.[32] OASIS-C2 is the current version of the OASIS data set and was implemented in 2017 in order to comply with requirements for standardized, cross setting measures for postacute care under the Improving Medicare Post-Acute Care Transformation (IMPACT) Act of 2014. Major items on the OASIS-C2 include sociodemographic, environmental, support system, health, and functional status. Based on these assessments, a care plan can be generated. The OASIS-C2 document collects detailed information on all pressure injuries as well as more limited information on stasis ulcers and surgical wounds. OASIS-C2 assessments are completed on admission to home health, at 60 days, on discharge, and with any hospitalization.

Data from OASIS-C2 are used as part of the Home Health Quality Reporting Program.[33] Both process and outcome measures are calculated. Process measures focus on the use of specific evidence-based activities such as routine immunizations and use of risk assessment tools. Outcome measures are all risk-adjusted and focus on rates of improvement in important areas such as activities of daily living, potentially avoidable events such as falls with injury, and utilization measures.

Accurate assessment of OASIS-C2 items by clinicians is essential. A number of studies have evaluated the reliability and validity of OASIS-C2 and have reported values in the low to moderate range, depending on the specific items.[34] These include assessments of interrater reliability in which the assessments of two different people are compared. Proper training of staff may help improve reliability and validity. Home health agencies are required to transmit OASIS-C2 data electronically to their state system. Improper

completion of OASIS-C2 can also lead to significantly lower payments or no payments at all. Thus, accurate assessments and charting are essential for recouping payments.

Physician Offices/Qualified Health Practitioners

The Medicare program pays for physician and other qualified healthcare practitioner services based on the Medicare Part B Physician Fee Schedule. Under this schedule, physicians and other qualified healthcare practitioners are paid for each medically necessary (and documented) service and procedure they perform. This includes office visits, surgical procedures, and a broad range of other diagnostic and therapeutic services. The Physician Fee Schedule is a list of 7,400 unique covered services and their payment rates. All services—surgical and nonsurgical—are classified and reported to CMS on claims according to the HCPCS code. When determining rates for each service, CMS considers such factors as the amount of work required to provide the service, practice expenses (the expenses related to maintaining a practice), and liability insurance. Through a calculation taking into account geographic variations, etc., Medicare pays the provider 80% of the fee schedule amount, and the Medicare beneficiary is liable for the remaining 20%.

Quality Improvement Efforts

Regulations related to reimbursements are tightly integrated with efforts in quality assessment and improvement. Indeed, care that is found not to meet quality standards may not be reimbursed. Even appropriate care may not be reimbursed if the condition being treated is the result of a medical error. Moreover, claims for reimbursements for substandard care could be viewed as fraudulent and result in criminal penalties. CMS does not rely solely on such punitive methods and various other initiatives exist. Most of these efforts center on pressure injuries, which may serve as a future model for other wounds. There are at least four ways in which quality measurement can be used to improve care, and different healthcare settings employ different approaches.

First, quality measurement is being used to empower consumers of health care. The assumption is that patients and their families, if given information about quality of care, will select those providers offering the best care. Such information then needs to be made available to patients in a timely fashion. Further, providers need to proactively improve their care in order to attract patients. This approach is exemplified by the Home Health Compare and Nursing Home Compare websites maintained by CMS. These sites contain not only facility rates of performance but also national and statewide rates to permit comparisons. To further facilitate use of this information by consumers, Nursing Home Compare employs a five-star rating system that combines information on these quality measures with results from state surveys and staffing levels. Whether this approach will indeed be successful in improving care, however, remains unknown.[35]

Second, quality measures are being used in quality improvement activities (IAs). The systematic use of such data can aid in the identification of quality-of-care problems and help determine the nature of these problems.[36] Nearly all healthcare provider organizations are involved in continuous quality IAs, with varying levels of implementation into clinical practice. A central component of such activities is feedback on performance. Indeed, demonstration

projects have suggested that providing home care agencies with performance feedback does result in reduced rates of hospitalization.[35]

Third, quality measures may help to focus more detailed analyses of the care provided to individual patients. Patients flagged by the Hospital Compare indicator may undergo a more detailed review of the care processes associated with the development and treatment of a pressure ulcer/injury. In nursing homes, state survey agencies are required to conduct annual unannounced surveys at SNFs to determine compliance with federal regulations regarding quality of care. A major focus of these surveys is an evaluation of pressure ulcer/injury prevention and treatment practices and whether the SNF is compliant with care.[37] Cases reviewed are often identified based on the MDS quality indicators.

Finally, CMS is increasingly relying on pay for performance and value-based purchasing as an important way of using reimbursements to improve care. Providers delivering the best care will be reimbursed more than providers delivering poor-quality care. While in theory this should be a highly effective mechanism for quality improvement, the data to date, which do not involve wound care, have not been convincing.[38] Basic issues such as the appropriate dollar amount to incentivize care, whether pay for performance represents a reward or an agent of change, and how best to measure care have not been completely resolved. While a number of projects have evaluated pay for performance in hospital and ambulatory care settings, demonstration projects involving nursing homes are in early stages. The extent to which pay for performance will focus on wound care is uncertain.

Role of Quality Measurement

Measuring quality is central to ensuring quality care. If you don't measure quality, you can't improve it. Facilitating such quality measurement is the wealth of data available in existing CMS databases, such as MDS 3.0 and OASIS-C, which provide patient-specific information on processes and outcomes of care. ICD-10 codes from hospital stays are also now much more informative. These changes may address some of the problems that have been identified in the past when using ICD-9-CM codes to measure rates of pressure injuries in hospitals.[39] Undercoding and misstaging of pressure injuries seem to remain a concern.[40,41]

Using these data sources, CMS is disseminating quality measures specific to different healthcare settings. In nursing homes, CMS reports two measures on its Nursing Home Compare website: the percent of short-stay residents with pressure ulcers/injuries that are new or worsened and the percent of long-stay high-risk residents with pressure injuries. Measures are being standardized across settings. Nursing Home Compare, Long-Term Care Hospital Compare, and Inpatient Rehabilitation Facility Compare all report on percent of pressure injuries that are new or worsened. The Hospital Compare website includes pressure ulcer/injury rates among its complications of care. While these measures of quality of care are widely available on CMS websites, their use has not been free of criticism. One issue has been the quality of the data, particularly ICD codes. Pressure injury rates calculated from clinical data are 10-fold higher than rates based on ICD-9-CM codes.[16,42] Not surprisingly, the positive predictive value of the hospital pressure injury rate based on these ICD-9-CM code is limited, although this may be less of an issue since the implementation of the POA code and ICD-10 CM.[15] A second issue is whether these rates should be adjusted for residents' risk of developing a new or worsening pressure injury. Studies have shown that differences in facility performance mostly represent differences in resident/patient mix rather than in facility performance.[43] Finally, these rates may not capture true differences in performance. Nursing homes that performed well and poorly on the pressure injury measure

were found to have few differences in how care was actually delivered.[44] Another study in 52 nursing homes found that a successful quality improvement effort was not associated with an improvement in the publicly reported CMS quality measure, but there was a decline in the incidence of stage 3 or 4 pressure injuries.[45] This again suggests that the CMS quality measures may not always capture differences in care performance.

Many other measures of wound care quality exist. Organizations such as the National Quality Forum will review these measures and determine whether they meet specific standards for reliability and validity. Those measures meeting these standards can be found on their website. One example is the Assessing Care of Vulnerable Elders (ACOVE) indicators for pressure injuries, which consist of a set of 11 indicators that capture different aspects of pressure injury care.[46] Each indicator is structured as an *if–then* statement, where the *if* component specifies a specific situation and the *then* component indicates what should be done in that situation.

Quality Measures and Qualified Health Professionals

On January 1, 2017, CMS began a new method for determining Medicare payments to qualified health professionals (QHPs) like physicians and nurse practitioners. Most QHPs are subject to the MIPS, which assigns a score from 0 to 100 for each QHP based on four performance categories, which include the reporting of quality measure data (QMs), clinical practice IAs, promoting interoperability of electronic health data, and cost. The composite score of these four weighted categories determines whether the QHP simply retains their Medicare Part B billing or experiences a bonus or penalty. MIPS is a zero-sum game, so bonus money is taken from the contributions of physicians who experienced penalties. The goal, over time, is to gradually increase the importance of the cost component. Because so many practitioners were exempted for various reasons during Year 1, the maximum bonus for a perfect score of 100 was less than 2%, which significantly reduced the willingness of practitioners to maximize their participation for a bonus. In 2019, a -7% penalty is possible, so practitioners are motivated to achieve at least the minimum points to avoid that.

The likelihood of succeeding at MIPS is significantly increased if QHPs participate through a Qualified Clinical Data Registry (QCDR). CMS has approved the U.S. Wound Registry (USWR) as a MIPS registry. A complete list of the USWR quality measures[47] can be found on the USWR website.[48] The barrier to reporting wound care–relevant QMs has been the reluctance of EHR vendors to incorporate the programmatic language for the QMs. The absence of significant bonus money has made this an insurmountable barrier although data are accumulating that practitioners who report even basic measures like diabetic foot ulcer (DFU), off-loading, venous leg ulcer (VLU) compression, and arterial screening have at least a 10% improvement in the healing rate of those respective ulcers. Benchmark rates have been set for DFU and VLU healing using the Wound Healing Index, which levels the playing field so that practitioners caring for more

severe ulcers are not penalized by making their outcomes look worse than their peers. The WHI allows the impact of quality improvements to be evident. The USWR has developed clinical practice IAs relevant to wound care. The USWR IAs may provide a way for practitioners unable to report wound care quality measures to still participate in wound care–related activities and achieve the minimum MIPS score to avoid a penalty.

Managing and Improving Care

Beyond these nonspecific approaches, CMS also actively promotes quality IAs directed toward Medicare beneficiaries. The primary mechanism for this is through quality improvement organizations (QIOs). QIOs are nongovernmental organizations, usually working under contracts, that aim to implement evidence-based practices through structured learning and collaborative efforts. They frequently focus on targeted medical conditions and priority populations in their efforts to improve patient safety and clinical outcomes.

In wound care, most of these efforts have again centered on pressure injuries. In New York, toolkits have been developed with which hospitals can assess and improve their pressure injury prevention and treatment practices. In nursing homes, QIOs from three states developed a strategy to train nursing home teams in quality improvement methods and proper pressure injury care. This training was reinforced through the use of outside mentors who regularly met with the teams. As a result of these initiatives and interventions, key processes of care improved dramatically.[49] A particularly impressive quality improvement collaboration within the New Jersey Hospital Association that involved over 150 hospitals and nursing homes resulted in reduction of more than 70% in pressure injury rates statewide.[50]

Government agencies other than CMS are also promoting initiatives to improve skin care, although in a nonregulatory manner. The Agency for Healthcare Research and Quality (AHRQ) has developed a toolkit for preventing pressure injuries among hospital patients.[51] This toolkit is unique in the strong emphasis it places on evaluating organizational readiness for implementing changes in practice. For nursing homes, AHRQ has recently promoted a clinical decision support instrument that has been shown to lead to significant reductions in pressure injury incidence.[52]

Results of Improvement Efforts

Evidence is demonstrating that these efforts spearheaded by CMS and other healthcare organizations are having an impact on improving pressure injury preventive care. Many hospitals have significantly reduced their rates of pressure injury occurrence. As one example, the Collaborative Alliance for Nursing Outcomes (CALNOC), a registry of 78 hospitals mostly in California, demonstrated a decline in hospital-acquired pressure injuries from 10.4% to 1.8% between 2003 and 2010.[53]

Summary

Regulatory agencies play a major role in wound care. With the increasing need to evaluate the cost-effectiveness of wound care, regulatory agencies will likely impose further regulations, which will lead to greater complexity in obtaining and maintaining reimbursements. Thus, the key to providing optimum wound care will depend on good documentation that clearly articulates the need for services and products and clearly identifies assessment of the patient, interventions instituted, and outcomes achieved. When this is accomplished, the patient, the provider, and the regulatory agency all benefit.

PATIENT SCENARIO

Clinical Data

Mr. Y, a 72-year-old resident from a long-term care facility, is admitted to the hospital for treatment of pneumonia. He was receiving treatment for a stage 3 pressure injury on his sacrum at the long-term care facility. There is no documentation about the injury by the physician in the hospital admission medical record. The nursing admission record documents the presence of a stage 3 pressure injury on the sacrum. Mr. Y is treated successfully for his pneumonia and is returned to the long-term care facility.

Case Discussion

The financial implications regarding use of POA coding have been in effect since October 1, 2008. Under CMS ruling, the practitioner responsible for establishing the medical diagnosis needs to document the diagnosis on admission. In this case, the POA pressure injury was not documented by the physician; therefore, the hospital was poised to lose a higher amount of reimbursement for the DRG of a stage 3 pressure injury as a secondary diagnosis. The hospital coder noticed the difference between the physician and

nursing documentation and queried the physician. Once it was established that the pressure injury was indeed POA, the physician completed his progress note and documented the location and stage of the pressure injury. The coder could then submit this secondary diagnosis for billing.

Show What You Know

1. **Medicare Part B is a federal program that:**
   A. supports state programs to provide services and products to the poor.
   B. reimburses hospitals for wound care services.
   C. reimburses for selected wound services and products in SNFs and home health agencies.
   D. doesn't require copayment from the beneficiary.

2. **For which one of the following healthcare settings is completion of OASIS-C2 required?**
   A. Hospitals
   B. Home health agencies
   C. Hospital outpatient centers
   D. SNFs

3. **Which one of the following criteria must a patient with a wound meet in order to qualify for SNF care?**
   A. Skilled services must be required for the same or related health problem that resulted in the hospitalization.
   B. The beneficiary must be in the hospital for 2 consecutive days.
   C. Services are needed once per week.
   D. The beneficiary must be admitted to the SNF within 90 days of admission to the hospital.

4. **Which of the following approaches *is not* being used by CMS to improve the quality of care?**
   A. Empower consumers to select high-quality providers through the provision of information on performance.
   B. Increase payments to providers of better care.
   C. Develop computer reminders on when to turn patients.
   D. Work with providers through regional QIOs.

Answers to these questions are found on page 681.

References

1. Centers for Medicare and Medicaid Services. Overview. 2010. Available at: http://www.cms.hhs.gov/History/. Accessed November 2, 2010.
2. Centers for Medicare & Medicaid Services. 2016 Medicare Enrollment Section, CMS.gov. June 14, 2018. Retrieved January 13, 2019 from https://www.cms.gov/Research-Statistics-Data-and-Systems/Statistics-Trends-and-Reports/CMSProgramStatistics/2016/Downloads/MDCR_ENROLL_AB/2016_CPS_MDCR_ENROLL_AB_2.pdf
3. Centers for Medicare and Medicaid Services. National Health Expenditures 2017 Highlights. Retrieved January 16, 2019 from https://www.cms.gov/research-statistics-data-and-systems/statistics-trends-and-reports/nationalhealthexpenddata/downloads/highlights.pdf
4. Centers for Medicare and Medicaid Services. 2006 Medicare Trustees Report [Press Release]. 2006. Available at: http:\\www.cms.hhs.gov/apps/media/press/release.asp?Counter=1846. Accessed November 2, 2010.
5. Medicare.gov. Part B Costs. January 2019. Available at: https://www.medicare.gov/your-medicare-costs/part-b-costs. Accessed January 28, 2019.
6. Centers for Medicare and Medicaid Services. Health Plans—General Information. September 2019. Available at: https://www.cms.gov/Medicare/Health-Plans/HealthPlansGenInfo/. Accessed January 28, 2019.
7. Nussbaum, S.R., Carter, M.J., Fife, C.E., et al. An Economic Evaluation of the Impact, Cost, and Medicare Policy Implications of Chronic Nonhealing Wounds. Value in Health 21:27-32, 2018. Available at: http://www.valueinhealthjournal.com/article/S1098-3015(17)30329-7/pdf
8. Centers for Medicare and Medicaid Services. Available at: https://www.cms.gov/medicare-coverage-database/overview-and-quick-search.aspx. Accessed January 20, 2019.
9. Hussey, P.S., Liu, J.L., White, C. “The Medicare Access and CHIP Reauthorization Act: Effects On Medicare Payment Policy and Spending.” Health Affairs 36(4):697-705, 2017. Available at: https://www.healthaffairs.org/doi/10.1377/hlthaff.2016.0559. Accessed on January 20, 2019.
10. Health Care Payment Learning & Action Network. Adoption of Alternative Payment Models Continues to Increase with 34% of Payments Tied to Value-Based Care. October 22, 2018. Available at: http://hcp-lan.org/workproducts/2018-APM-Progress-Press-Release.pdf. Accessed January 20, 2019.

Hess, C.T. “Clinical Quality Measures and Meaningful Use.” Advances in Skin
11. & Wound Care 27(5):240, 2014.
12. Centers for Medicare and Medicaid Services. License Agreements. 2018. Available at: https://www.cms.gov/medicare-coverage-database/details/article-details.aspx?articleId=55426&ver=50&LCDId=33831&CoverageSelection=Local&ArticleType=All&PolicyType=Final&s=All&KeyWord=surgical+dressings&KeyWordLookUp=Title&KeyWordSearchType=And&bc=gAAAACABAAAA&. Accessed January 18, 2019.
13. Medicare Payment Advisory Commission. Hospital Acute Inpatient Services Payment System. Available at: http://www.medpac.gov/docs/default-source/payment-basics/medpac_payment_basics_17_hospital_final65a311adfa9c665e80adff00009edf9c.pdf?sfvrsn=0. Accessed January 22, 2019.
14. Centers for Medicare and Medicaid Services. The Hospital Value-Based Purchasing (VBP) Program. Available at: https://www.cms.gov/Medicare/Quality-Initiatives-Patient-Assessment-Instruments/Value-Based-Programs/HVBP/Hospital-Value-Based-Purchasing.html. Accessed January 22, 2019.
15. Rosen, A.K., et al. “Validating the Patient Safety Indicators in the Veterans Health Administration: Do They Accurately Identify True Safety Events?.” Medical Care 50(1):74-85, 2012.
16. Smith, S., et al. Success in Hospital-Acquired Pressure Ulcer Prevention: A Tale in Two Data Sets. Health Affairs 37(11):1787-96, 2018.
17. Meddings, J.A., et al. Under Pressure: Financial Impact of the Hospital-Acquired Conditions Initiative-A Statewide Analysis of Pressure Ulcer Development and Payment. Journal of the American Geriatrics Society 63(7):1407-12, 2015.
18. Centers for Medicare and Medicaid Services. Hospital Outpatient PPS: Overview. 2010. Available at: https://www.cms.gov/HospitalOutpatientPPS/. Accessed November 2, 2010.
19. Centers for Medicare and Medicaid Services. Skilled Nursing Facility (SNT) Care. Available at: https://www.medicare.gov/coverage/skilled-nursing-facility-snf-care. Accessed January 15, 2019.
20. Schaum, K.D. “Medicare Payment: Surgical Dressings and Topical Wound Care Products.” Advances in Wound Care 3(8):553-60, 2014.
21. Centers for Medicare and Medicaid Services. Medicare and Medicaid Programs: Reform of Requirements for Long-Term Care Facilities. Available at: https://www.cms.gov/medicare/Provider-enrollment-and-certification/guidanceforLawsandRegulations/Nursing-Homes.html. Accessed January 15, 2019.
22. Saliba, D., Buchanan, J. Making the Investment Count: Revision of the Minimum Data Set for Nursing Homes, MDS 3.0. Journal of the American

Medical Directors Association 13(7):602-10, 2012.
23. Levine, J.M., Roberson, S., Ayello, E.A. “Essentials of MDS 3.0 Section M: Skin Conditions.” Advances in Skin & Wound Care 23(6):273-84, 2010.
24. Ayello, E.A. CMS MDS 3.0 Section M Skin Conditions in Long-Term Care: Pressure Ulcers, Skin Tears, and Moisture-Associated Skin Damage Data Update. Advances in Skin & Wound Care 30(9): 415-29, 2017.
25. Ayello, E.A., Levine, J.M., Roberson, S. “Late breaking CMS Update on MDS 3.0 Section M: Skin Conditions—Changes in Coding of Blister Pressure Ulcers.” Advances in Skin & Wound Care 23(9): 394-97, 2010.
26. Fries, B.E., et al. “Refining a Case-Mix Measure for Nursing Homes: Resource Utilization Groups (RUG-III).” Medical Care 32(7):668-85, July 1994.
27. Rantz, M.J., et al. “The Minimum Data Set: No Longer Just for Clinical Assessment.” The Annals of Long Term Care 7(9):354-60, September 1999.
28. Centers for Medicare and Medicaid Services. Medicare Issues Fiscal Year 2019 Payment & Policy Changes for Skilled Nursing Facilities. Available at: https://www.cms.gov/newsroom/fact-sheets/medicare-issues-fiscal-year-2019-payment-policy-changes-skilled-nursing-facilities. Accessed January 15, 2019.
29. Centers for Medicare and Medicaid Services. Home Health Prospective Payment System. Available at: https://www.cms.gov/Outreach-and-Education/Medicare-Learning-Network-MLN/MLNProducts/Downloads/Home-Health-PPS-Fact-Sheet-ICN006816.pdf. Accessed December 28, 2018.
30. Centers for Medicare and Medicaid Services. Clarification and Payment Policies for Negative Pressure Wound Therapy (NPWT) Using a Disposable Device. September 20, 2017. Available at: https://www.cms.gov/Outreach-and-Education/Medicare-Learning-Network-MLN/MLNMattersArticles/Downloads/SE17027.pdf. Accessed January 18, 2019.
31. Centers for Medicare and Medicaid Services. CMS Takes Action to Modernize Medicare Home Health. Available at: https://www.cms.gov/newsroom/press-releases/cms-takes-action-modernize-medicare-home-health. Accessed December 28, 2018.
32. Centers for Medicare and Medicaid Services. Outcome and Assessment Information Set (OASIS). Available at: https://www.cms.gov/Medicare/Quality-Initiatives-Patient-Assessment-Instruments/OASIS/index.html. Accessed January 2, 2019.
33. Centers for Medicare and Medicaid Services. Home Health Quality Measures. Available at: https://www.cms.gov/Medicare/Quality-Initiatives-Patient-Assessment-Instruments/HomeHealthQualityInits/Home-Health-Quality-Measures.html. Accessed January 2, 2019.

34. O'Connor, M., Davitt, J.K. The Outcome and Assessment Information Set (OASIS): A Review of Reliability and Validity. Home Health Care Services Quarterly 31(4):267-301, 2012.
35. Mukamel, D.B., Spector, W.B. "Quality Report Cards and Nursing Home Quality." Gerontologist 43(special issue II):58-66, 2003.
36. Karon, S.L., Zimmerman, D.R. "Using Indicators to Structure Quality Improvement Initiatives in Long-term Care." Quality Management in Health Care 4(3):54-66, 1996.
37. Lyder, C. "Pressure Ulcers in Long-Term Care: CMS Initiatives." ECPN 1:18-2, January 2005.
38. Petersen, L.A., et al. "Does Pay-for-Performance Improve the Quality of Health Care?" Annals of Internal Medicine 145(3):265-72, 2006.
39. Polancich, S., Restrepo, E., Prosser, J. "Cautious Use of Administrative Data for Decubitus Ulcer Outcome Reporting." American Journal of Medical Quality 21(4):262-68, 2010.
40. Squitieri, L., Ganz, D.A., Mangione, C.M., et al. Consistency of Pressure Injury Documentation Across Interfacility Transfers. BMJ Quality & Safety 27: 182-9, 2018.
41. Coomer, N.M., McCall, N.T. Examination of the Accuracy of Coding Hospital-Acquired Pressure Ulcer Stages. Medicare & Medicaid Research Review 3(4), 2013.
42. Meddings, J.A., et al. "Hospital Report Cards for Hospital-Acquired Pressure Ulcers: How Good are the Grades?" Annals of Internal Medicine 159(8): 505-13, 2014.
43. Li, Y., et al. "The Nursing Home Compare Measure of Urinary/Fecal Incontinence: Cross- Sectional Variation, Stability Over Time, and the Impact of Case Mix." Health Services Research 45(1):79-97, 2010.
44. Bates-Jensen, B.M., Cadogan, M., Osterweil, D., et al. The Minimum Data Set Pressure Ulcer Indicator: Does It Reflect Differences in Care Processes Related to Pressure Ulcer Prevention and Treatment in Nursing Homes. Journal of the American Geriatrics Society 51(9):1203-12, 2003.
45. Lynn, J., et al. "Collaborative Clinical Quality Improvement for Pressure Ulcers in Nursing Homes." Journal of the American Geriatrics Society 55(10):1663-669, 2007.
46. Bates-Jensen, B.M. "Quality Indicators for Prevention and Management of Pressure Ulcers in Vulnerable Elders." Annals of Internal Medicine 135(8 Part 2):744-51, 2001.
47. U.S. Wound Registry. MIPS Program Quality Measures. 2019. Available at: https://www.uswoundregistry.com/QualityMeasures. Accessed January 27, 2019.
48. U.S. Wound Registry. 2019. Available at: https://www.uswoundregistry.com/Home. Accessed January 27, 2019.

49. Baier, R.R., et al. “Quality Improvement for Pressure Ulcer Care in the Nursing Home Setting: The Northeast Pressure Ulcer Project.” Journal of the American Medical Directors Association 4(6):291-301, November-December 2003.
50. Holmes, A., Edelstein, T. “Envisioning a World Without Pressure Ulcers.” Extended Care Product News 122:24-9, 2007.
51. Berlowitz, D., et al. Preventing Pressure Ulcers in Hospitals: A Toolkit for Improving Quality of Care. Agency for Healthcare Quality and Research, 2011. Available at: http://www.ahrq.gov/research/ltc/pressureulcertoolkit/
52. Olsho, L.E., et al. “Evaluation of AHRQ’s On-Time Pressure Ulcer Prevention Program.” Medical Care 52(3):258-66, 2014.
53. Stotts, N.A., et al. “Eliminating Hospital-Acquired Pressure Ulcers: Within Our Reach.” Advances in Skin & Wound Care 26(1):13-8, 2013.

3

Legal Aspects of Wound Care

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We gratefully acknowledge the contributions of Gregory Brown, RN, ET, for his work on previous editions of this chapter.

Objectives

After completing this chapter, you'll be able to:

- explain the major litigation players and their roles in a lawsuit
- define the four elements of a malpractice claim
- describe the general rules for proper wound care charting
- indicate ways the medical record, standards, and guidelines can be used in a malpractice case
- describe documentation practices that predispose the medical record to legal risks
- describe strategies to improve consistency and accuracy of medical record documentation that minimize potential litigation risk.

The Current Climate

In recent years, the concept of patients as “consumers of health care” has risen to the forefront. Rather than blindly trusting clinicians, the consumer–patients of today are better educated, more aware of healthcare issues, and more willing to make use of legal resources when treatment goes awry. Easy access to the Internet has provided yet another information portal for consumer–patients to review writings about health care and disease states (including treatments that may or may not be evidence based) that can affect their views of the care they have received. Social media sites such as Facebook provide access to “pages” devoted to the experiences, many negative, of patients who underwent or are currently undergoing treatment that may also influence consumer–patients. Although wound care generates no more litigation than many areas of healthcare practice, and arguably less than some others, the threat of litigation may affect the way clinicians approach the delivery of care. A wound is “visible” to the patient and/or family, whereas failure of other organs is less or not at all “visible.”

Clinicians need to protect themselves while ensuring evidence-based, high-quality care to their consumer–patients. This chapter sets forth basic legal principles and suggests practice strategies that advance patient care *and* protect clinicians.

Litigation

During the course of human history, it became apparent that nonviolent means of settling disputes must be developed. The law and the legal process, including litigation, continue to be one of civilized society's experiments at achieving nonviolent resolutions to disputes. The success of this experiment is itself the source of much dispute, to which no resolution (nonviolent or otherwise) is currently in sight.

Contrary to television and film portrayals, the real-life litigation process is arduous and time-consuming. While fictitious television and film lawsuits resolve in a matter of weeks or months, usually ending with a dramatic trial resulting in a stunning jury verdict, most real-life cases take years to get through the legal system. In some jurisdictions with crowded dockets, and due to their complexity, medical negligence/malpractice lawsuits can take as long as 5 years to resolve. Those that require appeals can take considerably more time before all issues are finally put to rest. Trials (dramatic or not) are few and far between, as nearly all lawsuits are settled before trial. When trials do happen, they are usually slow-moving, uninteresting events that tax the patience and attention of jurors. Litigants expecting "Perry Mason" moments from their attorneys are sure to be disappointed, and, as anyone who has ever served on a jury knows, closing arguments by attorneys is never, ever over in the 5 minutes before the final commercial.

Despite the difficulties and drawbacks, the litigation process does afford citizens an impartial forum for dispute resolution grounded in the law. And the law, as Plato stated, is "a pledge that citizens of a state will do justice to one another."

The discussion in this chapter is limited to *civil litigation*, that is, litigation in which citizens have a dispute with each other—rather than *criminal litigation*, in which the state or a government seeks to prosecute a party for the violation of law. There are significant differences between the two forms of litigation (e.g., standards of proof). The remedy sought in civil litigation is monetary damages. In

contrast, only the prosecuting state or government may seek to deprive the alleged lawbreaker of his or her liberty by incarceration.

How Is a Medical Malpractice Lawsuit Born?

Litigation begins the moment a person believes he or she has been wronged by another and seeks the advice and counsel of an attorney in an effort to “right the wrong” or “get justice.” During the initial interview between the prospective client and the attorney, the attorney makes a number of preliminary judgments usually based solely on the client’s presentation:

- Is this the type of case the attorney is capable of handling? Does it fall within his or her expertise and practice experience? Does the attorney have the time to handle the matter?
- Is the client’s story credible?
- Will the client make a good witness?
- Are the damages, if proven, sufficient to warrant entering into the litigation process?
- Is there a party responsible (liable) for the client’s injuries?
- How likely it is that both liability and damages can be proven?
- Are there any glaring problems or difficulties with the case?

If the answers to these questions are satisfactory and the client wishes to retain the attorney, a lawsuit has then been conceived.

Before filing the legal documents that start the litigation process in a medical malpractice case, most attorneys perform an intensive investigation in order to definitively answer questions concerning liability and damages. Medical records and other information must be obtained and examined by an expert to determine whether a malpractice claim can be made. Information related to the identities of potential defendants must be analyzed, and strategic legal issues related to jurisdiction (which court can the case be brought in) must be thought through. If after this investigation the attorney still believes the case has merit, legal papers starting the actual lawsuit will be filed, and a lawsuit will be born (Box 3-1).

Box 3-1 Players in the Litigation Process

The litigation process is initiated and enacted by people with a dispute to resolve and those whose task it is to aid in resolving that dispute.

The Parties

The principal parties involved in litigation are the *litigants*—the individuals on either side of the dispute. The *plaintiff* is the person who initiates the lawsuit and who claims he or she has suffered injury due to the actions of another. A lawsuit may be filed by multiple plaintiffs.

The plaintiff sues the *defendant*—the person(s) or organization(s) alleged to have injured the plaintiff by his/her or its actions. In most cases, the parties are individuals, but parties can be corporations, companies, partnerships, government agencies, or, in some cases, governments themselves.

The Judge

The *judge* is an individual, usually an attorney, who has been appointed or elected to oversee lawsuits on behalf of the state or government under whose jurisdiction the lawsuit is brought. The judge acts as referee during the pretrial phase of the case and decides legal issues that arise as the lawsuit progresses toward trial. In a trial, the judge's responsibility is *to interpret the law*.

The Jury

The *jury* is a panel of citizens chosen by the attorneys for the litigants to hear evidence in the case and render a decision or verdict. The jury's responsibility is *to determine the facts* in a trial. It's up to the jury to decide whether the plaintiff and his or her attorney proved their case, thereby rendering a decision about the defendant's liability and the amount of damages the defendant should pay to the plaintiff.

The Pretrial Litigation Process

The pretrial litigation process consists of several steps: complaint and answer, discovery, and motion practice.

Complaint and Answer

The initial legal paper that gives rise to a lawsuit is called the *complaint*. While procedural requirements vary between jurisdictions, generally the complaint is a document that sets out the claims made by the plaintiff against the defendant, the basis of the jurisdiction of the court, the legal theories under which the plaintiff is making the claims, and, in some jurisdictions, the amount of damages claimed. The complaint must allege sufficient facts to establish a *prima facie* (based on the first impression or true until proven otherwise) case. Defendants may and often do file an application to the court before answering the complaint, seeking to have the case dismissed by the court before it starts based on insufficient factual detail in the complaint.

If the case is not dismissed at the outset, or if the defendants choose not to make that application, the defendant must then file an *answer* within the permitted time that responds on a count-by-count basis to the plaintiff's complaint and that, depending again on jurisdictional rules, may also include claims against the plaintiff. These two basic *pleadings* initiate the formal lawsuit.

Discovery

Discovery is the process by which the parties find out the facts about each other, about the incidents that have given rise to the claims of malpractice alleged by the plaintiff, and the defenses to those claims asserted by the defendant. The law has provided discovery devices—procedural mechanisms by which the parties ask for and receive information. Demands are routinely made for documents and other tangible items related to the lawsuit's claims, for statements made by the parties to others, and for the identification of witnesses to the incidents. Then, pretrial testimony (*deposition or examination before trial [EBT]*) is taken of the parties and fact witnesses to the lawsuit. This testimony, while out of court, is sworn testimony transcribed by a certified court reporter and can be used for any purpose in the lawsuit, including for purposes of *impeachment*—the demonstration of prior untruthful or inaccurate testimony, or a challenge to the credibility of a witness—at trial.

Finally, *expert discovery*—information about the opinions of experts retained by the parties—is usually permitted. Experts are individuals accepted by the court to assist the finder of fact—the jury—in understanding issues that commonly fall outside of the experience of the typical juror. In medical malpractice cases, the plaintiff must prove that there was a deviation from the standard of care that resulted in an injury. Expert testimony related to the field of medicine, treatments, and standards of care at issue in the case is essential to successfully meet proof requirements for each element of a malpractice claim brought by a plaintiff. Likewise, the defense of such claims requires opposing expert testimony—in essence, an explanation by a credentialed individual supporting the actions taken by the defendant from which the claim of malpractice stems.

Motion Practice

Disputes over discovery often arise in the context of a lawsuit, and those disputes that can't be resolved by the parties require court intervention. Formal resolution of these disputes usually requires an application to the court—a *motion*—setting forth the dispute and the position of the party making the application (the moving party or *movant*) and requesting certain *relief* or results to be *ordered* by the court. Naturally, this requires a response from the other party—the *opposition*—that sets out the reasons why the court shouldn't grant the relief requested.

Some motions can be decided by the court *on the papers*, that is, without a formal oral presentation (*oral argument*) by the parties before the judge is assigned. More complicated motions, especially those seeking to eliminate or modify legal claims, almost always require argument before the presiding judge or court.

The Trial

While the vast majority of lawsuits settle before trial ("out-of-court settlements"), some cases do proceed to trial. Medical malpractice trials are almost without exception jury trials. Once it's determined that settlement isn't an option, a trial date is set and the attorneys begin to prepare. In federal jurisdictions and many state courts, litigants are required to prepare pretrial statements and submissions. They also disclose exhibit lists (materials and documents the attorneys anticipate they will use at trial). They may also designate deposition testimony to be read or, if the testimony was videotaped, to be shown at trial. The pretrial submission and disclosure process helps to ensure that the trial is as fair as possible and eliminates the possibility of "trial by ambush." Thus, the "Perry Mason" moments of television and film renown are relatively few and far between.

On the day of the trial, the attorneys for the parties proceed with jury selection. Each attorney tries to select jurors that he or she believes will decide in favor of (*find for*) his or her client. Procedurally, the jury selection process varies widely by jurisdiction. In some courts, the trial judge will take an active role by questioning the jurors. Other jurisdictions permit the attorneys to question jurors directly without court supervision, and the trial judge becomes involved only when a significant dispute arises. The fight over selection is then left to the attorneys. As you can imagine, jury selection in a jurisdiction with strong judicial control is a much briefer process than in those jurisdictions where the attorneys are left to their own devices. No matter what the individual procedure, once the jury is chosen (*empanelled*), the trial begins.

At trial, the parties each give an opening statement, one of the two times in the entire trial that the attorneys are permitted to speak directly to the jurors. After the opening statements, the plaintiff's attorney presents the plaintiff's case. As the burden of proof is on the plaintiff, the plaintiff's attorney goes first. After the plaintiff's direct case is finished, the plaintiff "rests," and the defendant's attorney presents the defendant's case. The *direct case* consists of evidence (testimony, documents, etc.) presented by the attorney for a party.

The party has the right to cross-examine each witness after the direct examination, and then additional examination may follow (“redirect” and “recross”) as necessary. After all the evidence has been presented by both sides, the parties make closing statements (*summations*), which is the last time the attorneys are permitted to speak directly to the jurors.

Once summations are completed, the judge then instructs the jurors on the appropriate law that they’re to apply to the facts of the case. Remember that the jury is the *finder of fact*—it determines what happened, when it happened, who did it, where it happened, and how it happened—and the judge is the *interpreter of the law*. After the jurors receive the judge’s instructions, they leave the courtroom and begin deliberations (Fig. 3-1).

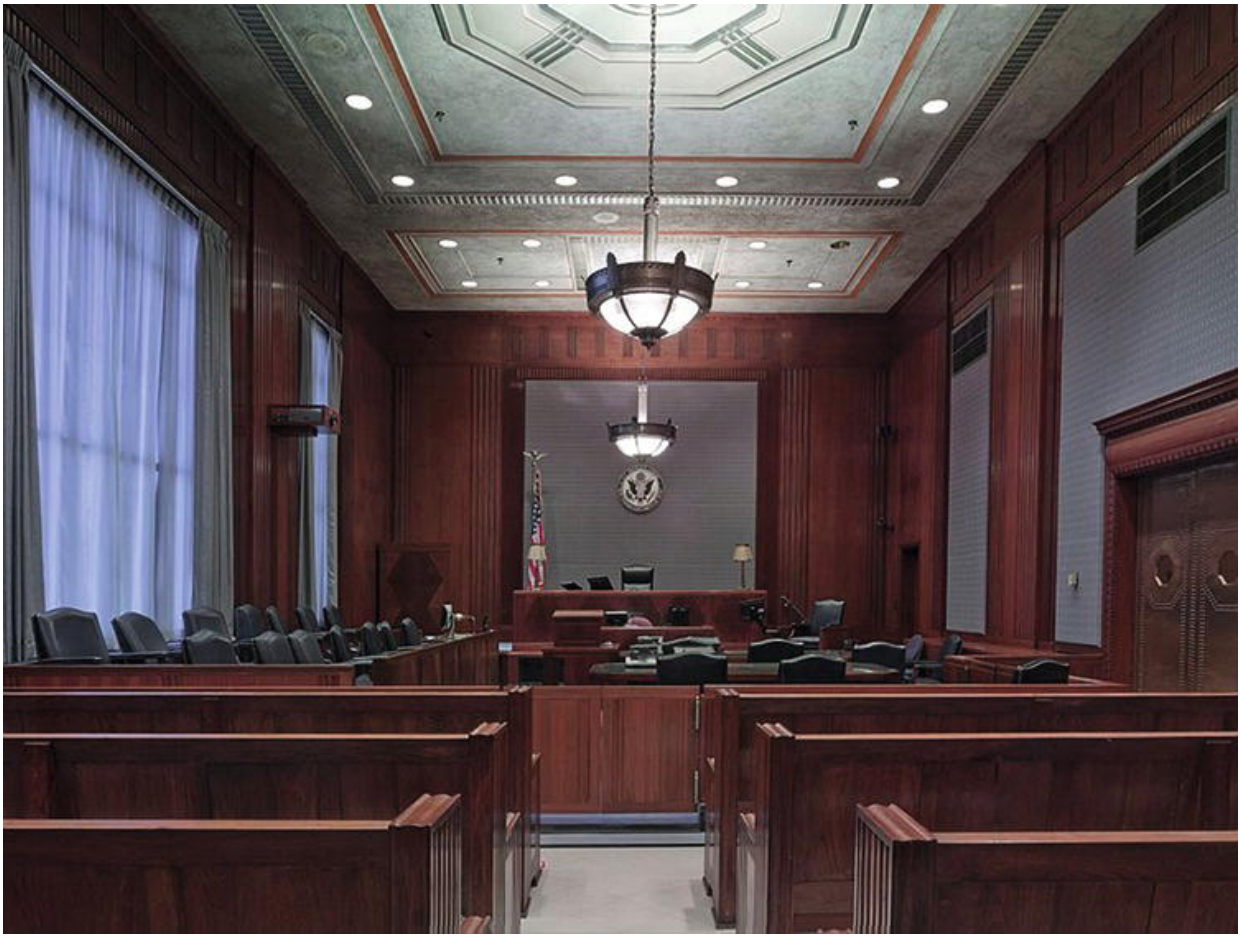

Figure 3-1 Courtroom.

Every trial attorney hopes to be lucky enough to serve on a jury that goes to deliberations. For trial lawyers, understanding what happens inside the jury room during deliberations is the Holy Grail of trial practice. In jurisdictions that permit attorneys to interview jurors after verdict, attorneys often spend many hours with the jurors who are willing to discuss the case in order to determine what did—and what didn't—work during the trial. It's often surprising to find that what the lawyer thought was of prime importance wasn't so important to the jury. The jury room in our legal system is sacrosanct, and, no matter how it happens, the jury will arrive at a verdict that will be delivered to the parties in open court. Once the verdict is read and the jury excused, the trial is over.

Appeals

Each jurisdiction has an appellate process, of which the litigants may take advantage. Depending on the jurisdiction, appeals may add years (and many dollars) to the resolution of claims and lawsuits.

Legal Elements of a Malpractice Claim

A medical malpractice claim is made up of four distinct elements, each of which must be proven to the applicable standard of proof in the jurisdiction of the case. The usual standard of proof for civil cases is a *preponderance of the evidence*. The preponderance standard can be best described as a set of scales that represent the plaintiff on one side and the defendant on the other, which are evenly balanced at the start. The party that wins is the one on the side of the scale that dips lower at the end of the trial. In other words, in order to prevail, plaintiffs need to show by only 50.0000001%—just a bit more than one-half—that they've proven each of the elements that make up a malpractice claim.

The four general elements that make up a malpractice claim are:

- existence of a duty owed to the plaintiff by the defendant
- breach of that duty
- an injury that is causally related to that breach of duty
- damages flowing from that injury that are recognized by law.

Duty

In general, there is no duty to protect a person endangered by the actions or omissions of another if there is no special relationship between the two persons. The patient–physician relationship is the basis for the claim of duty between the plaintiff–patient and the defendant–healthcare professional in medical malpractice cases because that relationship requires the patient to rely on the physician's knowledge, expertise, and skill in treatment. Thus, the allegations of medical negligence arise within the course of that professional relationship. Translating that definition into healthcare terms, some examples of a duty may be the obligation of a healthcare practitioner to give patients care that is:

- consistent with the level of his or her experience, education, and training
- permitted under the applicable state practice act
- authorized or permitted under the policies and procedures of the institution that are applicable to the position.

Practice Point

Duty: In negligence cases, *duty* may be defined as obligation, to which the law will give recognition and effect, to conform to a particular standard of conduct toward another. The word *duty* is used in the law to denote the fact that the actor is required to conduct himself in a particular manner at the risk that if he doesn't do so, he becomes liable to another to whom the duty is owed for any injury sustained by such person, of which that actor's conduct is a legal cause (Restatement, Second, Torts, Section 4).[1]

Breach of Duty

In addition to proving the existence of a duty, the plaintiff must also prove the defendant breached that duty. Breach of duty can result from commission, omission, or both. Most often, to establish this element of the claim, the plaintiff in a medical malpractice case must also show that the defendant healthcare practitioner deviated from an accepted standard of care or treatment. In defense of the claim, the practitioner isn't required to provide the highest degree of care but only the level and type of care rendered by the *average practitioner*. What the standard of care is, and whether and how it was deviated from, must be established for the jury, and this is most often the province of expert testimony.

Breach of duty in the healthcare setting may be illustrated in the following ways:

- Failure to give care within the applicable practice act
- Failure to perform professional duties with the degree of skill mandated by the applicable practice act
- Failure to provide care for which the circumstance of the patient's condition warrants

Practice Point

Breach: The failure to meet an obligation to another person that's owed to that person; the breaking or violating of a law, right, obligation, engagement, or duty by commission, omission, or both.[1]

Injury Causally Related to a Breach of Duty

In a medical malpractice case, proof of an injury isn't enough to carry the day unless that injury can be causally linked to a breach of duty by a healthcare practitioner. That breach of duty is then considered the proximate cause. Without the breach of duty, the injury wouldn't have occurred (Box 3-2).

Box 3-2 Proving Proximate Cause

While standards of proof related to proximate cause may vary among jurisdictions, one of two questions is almost always used to determine this issue:

- Was the healthcare practitioner's negligent conduct a "substantial factor" in causing the injury?
- Would the injury not have happened if the healthcare practitioner hadn't been negligent?

Proximate cause in the healthcare setting can be illustrated by the following examples:

- Fractured hip due to a fall because of failure to raise the side rails of the bed
- Decreased total protein due to failure to provide nutrition (either failure to provide actual nourishment or failure to order/call a consult)
- Osteomyelitis resulting in limb amputation following failure to attain/call an infectious disease consult and provide antibiotic therapy

Practice Point

Proximate cause: That which, in a natural and continuous sequence, unbroken by any efficient intervening cause, produces injury, and without which the result wouldn't have occurred and without which the accident couldn't have happened, if the injury be one that might be reasonably anticipated or foreseen as a natural consequence of the wrongful act.[1]

Damages

Finally, the fourth element that makes up a malpractice claim is damages. A healthcare practitioner may be held liable for damages when the jury finds that the practitioner deviated from the applicable standard of care in treating the plaintiff–patient and, as a result, caused injury resulting in legally recognized damages. In most jurisdictions, a plaintiff may recover for proven monetary losses (lost wages and unreimbursed medical expenses) and for pain and suffering that result from the proven injury. As noted previously, it's the jury—the finder of fact—that sets the monetary award to the plaintiff.

Practice Point

Damage: Loss, injury, or deterioration caused by the negligence, design, or accident of one person or another, with respect to the latter's person or property.

Damages: A pecuniary compensation or indemnity that may be recovered in the courts by any person who has suffered loss, detriment, or injury, whether to his or her person, property, or rights, through the unlawful act or omission or negligence of another.[1]

As we have shown, in order for a plaintiff to prevail in a medical malpractice claim, *all four of the elements* discussed above must be satisfied. Three of four won't do. They must score perfectly on all four to prevail before a jury.

The Medical Record in Litigation

The medical record is arguably the single most important piece of evidence in a medical malpractice case. It serves as a crucial tool for the delivery of evidence-based/science-based care. It is also:

- a legal document
- a communication tool
- the supporting basis for treatment decisions and modifications
- one of the primary tools for the evaluation of treatment modalities.

At one time or another in the education of a healthcare practitioner, whatever the specialty or discipline, this directive is taught: “If it wasn’t written down, it didn’t happen.” Nowhere does this statement ring truer than in a medical malpractice case (Box 3-3). Before we consider the role documentation plays in the medicolegal world, let’s first consider for a moment how important the medical record is in the care and treatment of patients.

Box 3-3 Effects of Incomplete Charting (Newly Bulleted)

What happens when charting is incomplete?

- In addition to providing a poor medical record of a patient’s care to help jog the practitioner’s memory if a lawsuit occurs, it can create other problems.
- Nothing makes proving the plaintiff’s case easier than such gaps, especially near or around the time of the alleged malpractice if the claim revolves around a single incident.
- If the claim concerns a continuous or extended course of treatment, the absence of documentation related to treatment outcomes, observations, and the basis for the treatment will be presented at trial as strong evidence of negligence.

- Where the record contains gaps, you can be certain that the plaintiff's attorney will be able to suggest to a jury what happened during those undocumented times, and those suggestions won't be of benefit to the healthcare facility or the individual practitioner.

The medical record serves several purposes. First and foremost, it is a communication tool that allows real-time coordination of care by multiple disciplines. It also acts as a historical record to determine the efficacy of past interventions and guide future care. The medical record is also a factual record utilized in lawsuits to determine the quality of care rendered, the occurrence of physical harm, and other legal issues. Most state boards (nursing, medical, and other licensed healthcare professionals) and federal regulatory agencies require "timely and accurate" documentation of findings in the medical record. Due to the wide range of specialty care areas and ever-changing rules, regulations, and laws, state and federal boards offer little guidance on *how* to meet this documentation standard. It's left to the individual facility or provider to determine the appropriate standards. The absence of standards has resulted in a wide range of documentation practices.

Communication Tool

The medical record is the primary method of communication between members of the healthcare team. Oral report and rounding are essential communication devices, but it's impractical and unrealistic to expect that every healthcare team member be present during report or rounds. Such disciplines as physical therapy, occupational therapy, and respiratory therapy may not be present for report or rounds. The myriad medical specialists available to the primary physician (e.g., infectious disease consultants) are also rarely present during rounds, yet it's imperative for the delivery of good science-based care that every healthcare team member has the most current and up-to-date patient information. The medical record is the only way to accomplish this. It is available 24 hours a day to any practitioner caring for the patient, who can utilize it to stay informed about the patient's progress.

Treatment Evaluation and Support

Documenting patient treatment outcomes and responses in the medical record is a key method for evaluating treatment modalities and therapies. For example, the patient with pressure injuries will undergo an extended course of treatment that will change over the course of time. In order to establish a basis for initial treatment and treatment modifications, there must be well-documented observations and evaluations of the patient. Upon initiation of treatment, careful observation and documentation of the patient's condition is critical in order to establish a baseline for initial treatment and care. Without a carefully documented record, treatment, evaluation, patient outcomes, and treatment modifications are impossible to justify in court (Box 3-4).

Box 3-4 General Documentation Guidelines

Listed here are some general rules for documentation that serve your patient's needs and can help in the defense of a lawsuit.

- Be thorough—record the date and time for each entry.
- Be accurate—use units of measure instead of estimates (e.g., "patient had a 6-oz cup of ice chips" instead of "patient had some ice chips").
- Be factual—think of yourself as a newspaper reporter and answer the following questions: who, what, when, where, why, and how.
- Be objective—record only the facts. Remember that you're communicating information that others will rely on. If your patient is to benefit from your professional training, judgment, and observational skills, your colleagues must have objective, factual information to rely upon. Save your opinions and differences over treatment alternatives for the discussions with healthcare team members.
- Select appropriate descriptors and drop-down menus when using computerized documentation systems; follow facility policy regarding system/computer failure for

documentation. Write legibly for manual records, and print if necessary.

- Only use approved abbreviations and terminology.
- Make contemporaneous entries—finish your documentation before you leave work for the day. Don't add notations days later unless your facility permits such additions—and even then, adhere strictly to your facility's policy governing such additions.
- Be truthful—don't fake, misrepresent, exaggerate, or misstate the facts in the medical record.
- Most importantly, don't assign blame. While it's important to relate the facts completely and accurately, assigning blame in the medical record is fodder for malpractice actions and does nothing to advance the care of your patients.

Practice Point

Accurate and complete patient outcomes and responses to treatment and care must be documented in the record, as they're the basis for care decisions and legal defense.

Legal Aspects of Wound Documentation

Wound assessments (see Chapter 6, Wound Assessment) are some of the most detailed and time-consuming documentation a healthcare provider will perform. Radiologists can view internal organs with a variety of internal imaging techniques and generate detailed, consistent reports. Myriad laboratory values are also available to monitor internal organ system functions. Wound assessment and documentation are still mostly a subjective, visual, pen-and-paper/computerized exercise that requires a competent knowledge base to perform accurately. Wound assessment and monitoring are typically left to the staff nurse or wound specialist. Wounds require an intricate, multifaceted assessment of their many attributes. Differing levels of knowledge among caregivers can result in inaccurate, inconsistent, and erroneous wound documentation. Lack of established documentation policies/guidelines (including terminology) within an institution or the failure of caregivers to follow existing policies/guidelines can lead to inconsistent and inaccurate documentation. Wound documentation may be in multiple areas of the medical record making it difficult to locate all of the patient's wound information. Multiple wounds on a single patient add even more of a documentation burden. Such complexity can lead to inconsistent documentation—and treatment—and may leave a provider or facility open to legal liability.

The American Nurses Association[2] has clarified that nurses are expected to record their assessments and diagnoses of the patient's skin integrity in the medical record. Staging of pressure injuries and differentiating them from other wounds is within the scope of nursing practice.

We recommend the following processes and procedures to improve the consistency and documentation of care for pressure injuries, acute and chronic wounds, or any other untoward event that occurs while a patient is under care. These recommendations are not designed to promulgate or establish a particular standard of care for

wound documentation but rather to make providers aware of the common difficulties that may occur with wound documentation and to propose solutions.

The Flow of Information

To find the latest laboratory value in a chart, you go to the lab section. To find the latest chest x-ray result, you go to the radiology section. To find the latest wound description, you go to…? Wound documentation is often found scattered throughout the chart in activities of daily living (ADL) forms, nursing assessments, narrative notes, wound assessment forms, physician progress notes, and many other sections. The organization and composition of the medical record in any given facility often evolves over the years with no real appreciation for how one would look at the chart in its entirety. This is especially true of nursing documentation forms as they are added to and modified over time. In such situations, duplication of information becomes increasingly common. Consistent documentation by nursing staff then becomes much more difficult to provide and to review at a later date.

In order to evaluate the effectiveness of the medical record as a communication tool, one should critically examine from an outsider's perspective just how information flows within the record. A medicolegal reviewer attempting to determine if care was accurately and consistently provided can sometimes be stymied by a poorly structured chart whose organization makes sense to the facility—yet to no one else. Information that cannot be found readily (drop-down screens) will often be ignored. If the appropriate form or the proper location in the chart cannot be found easily, certain information may not be documented. If wound care documentation takes place in multiple areas of the chart, it may be documented multiple times—or not at all—because of the uncertainty as to where the information should be properly entered. The medical record is a documentation system. If documentation is inconsistent, a *systems approach* may be applied in order to evaluate and improve the structure and flow of documentation, rather than actually getting staff to just "document more."

Admission Assessment

Both the medical and nursing admission assessments provide a "snapshot" of the patient's status at the time of admission. The admission assessment is an area where one cannot over document. The more pertinent information documented about the patient at the time of admission, the better informed the healthcare team will be, and thus, decisions can be made based on the best information available. Discovery and description of any lesion during admission assessment are critical in determining the course of care and, in a lawsuit, in determining the liability for any wounds that develop or deteriorate during the patient's stay. Preexisting lesions should be documented carefully and thoroughly with regard to size, location, and characteristics. *A detailed description of the wound is more important than an actual wound diagnosis during admission assessment, as wounds have varying etiologies.* The chart should reflect what interventions were taken and who was notified of the existence of the wound and any other findings.

Since pressure injuries are a frequent reason for litigation in health care, they will be highlighted as a key exemplar in the remainder of this chapter. Often heard in nursing and wound care circles is that any pressure injury (PI) that develops after 24 hours of admission is considered to be acquired at the facility rather than inherited. However, not all skin changes may be visible on admission, for example, deep tissue injury (DTI) describes intact or nonintact skin with localized area of persistent nonblanchable deep red, maroon, purple discoloration or epidermal separation revealing a dark wound bed or blood-filled blister, as updated by the National Pressure Injury Advisory Panel (NPIAP) in 2016.[3] Pressure ulcer prevention and treatment guideline makes this general rule difficult to defend. For example, the patient may be admitted with an inconspicuous-looking skin discoloration in the sacral area or other bony prominence that ultimately evolves into a full-thickness pressure ulcer. Emerging research supports the process that tissue ischemia and necrosis can take several days to become visible to the naked eye when all that was observed and documented was a skin discoloration.[4,5]

Therefore, nursing, medical, and all license healthcare professional staff should be made aware of this NPIAP definition[3] and incorporate it into their wound assessments (for more information, see Chapter 14, Pressure Injuries).

Pressure Ulcer Risk Assessment

All patients should be assessed for PI risk. A PI risk assessment can include any validated scale, such as the Braden or Norton scale. Risk scales are tools that quantify risk factors associated with pressure ulcer development, such as nutrition, moisture, and mobility, among others, which should be used to establish a picture of the patient's health status on admission. This initial evaluation is crucial for proper care planning as well as defense of negligence claims. A PI risk assessment scale can provide detailed insight into the care needs of the patient far beyond skin protection. The frequency of risk assessment is open to debate and is based primarily on the guidelines or custom of the individual nursing unit.

Ideally, every patient admitted to a healthcare facility should be assessed upon admission to identify individuals at risk for PI development. Those at risk should then have routine follow-up assessments during their stay. A risk assessment is also recommended when the patient is transferred to another unit or whenever there is a significant change in the patient's condition. The nursing admission and/or daily assessment is a logical chart area in which to document risk assessment. The most important aspect with any risk assessment is: What is done with the information? Validated risk assessment tools are powerful and accurate predictors of PI development but are useless if the information they provide is not acted upon. Each risk factor is a data point to be evaluated in the formulation of an individual plan to prevent, mitigate, or improve a decline in the level of functioning.

Pressure Ulcer Development

Sometimes, the underlying problems that result in the development of PI can be managed, healed, or avoided. In other instances, the disease burden can be so great that ulcers will occur or fail to heal despite the best of care. Indeed, the Centers for Medicare and Medicaid Services (CMS) recognizes that pressure injuries are unavoidable in long-term care if the facility had[1] evaluated the resident's clinical condition and PI risk factors[2]; defined and implemented interventions that are consistent with resident needs, goals, and professional standards of practice[3]; monitored and evaluated the impact of the interventions; and[4] revised the approaches as appropriate.[6] In 2010, the NPIAP hosted a consensus conference and modified this definition to make it applicable to other care settings. The revised definition of "unavoidable" in reference to PI development is "that the individual developed a pressure ulcer even though the provider had evaluated the individual's clinical condition and pressure ulcer risk factors; defined and implemented interventions that are consistent with individual needs, goals and recognized standards of practice; monitored and evaluated the impact of the interventions; and revised the approaches as appropriate".[7] In 2014, the NPIAP had another consensus conference that affirmed this definition and reached consensus on several clinical conditions that support PI being unavoidable.[8]

Any documentation system regarding PI prevention must clearly and efficiently outline these criteria.

Practice Point

Consider the following NPIAP consensus conference statement that there are patient situations that could lead to unavoidable

pressure injuries[7,8]:

- skin failure
- hemodynamic instability that may preclude turning or repositioning
- patient refusal to reposition despite education on the rationale for doing so and the consequences thereof.

Nursing units where the disease burden of patients is extremely high include intensive care units (ICUs), long-term care units, and hospice. For ICU patients, frequent and consistent monitoring should be performed, particularly on the high-risk areas of the sacrum, heel and trochanter, and the occipital area. We frequently find in chart reviews that dynamic, pressure-distributing mattresses are obtained *after* the development of a PI. While this may be a logical and justifiable escalation of care and intervention for a general medical–surgical population, it's important that special emphasis be placed on these high-risk populations.

The Infamous “Turn Q 2” Check Box

Many nursing patient care flow sheets have “Turn Q 2” (turn every 2 hours) on their checklist of PI prevention strategies. The presence or lack of a check in this box on these flow sheets is often used by attorneys to undermine or paint a negative picture of the quality and consistency of care delivered by nursing staff. The origins of the requirement to reposition patients every 2 hours for PI prevention are obscure and not well grounded in science. The 1994 Agency for Health Care Policy and Research (AHCPR) guidelines specifically recommended repositioning every 2 hours.[9] Such an absolute time requirement for repositioning or other interventions does not permit individual clinical judgment. Patient care should be based on the dynamic evaluation of a patient’s status by qualified personnel and not on a single, fixed point. Some patients may require more frequent repositioning and some less as a result of the use of a pressure-distributing mattress[10,11] (which were rudimentary when the q2 hour recommendation was originated) or the need for uninterrupted sleep. Some may be unable to turn due to critical illness. Some may have undergone diagnostic procedures, thereby precluding staff from attending to them every 2 hours. There are simply too many variables that determine when and how a patient is positioned to require a rigid timetable that likely bears little or no resemblance to the patient’s actual needs. The NPIAP–European Pressure Ulcer Advisory Panel (EPUAP) prevention and treatment guideline states that repositioning frequency should be influenced by the support surface used (strength of evidence = A).[3]

We recommend removing the “Turn Q 2” check box from nursing forms, admission order templates, pressure ulcer prevention orders, and other areas. The check box could be replaced with a statement such as “reposition according to patient needs as determined by PI risk assessment” or some other language determined to better meet patient care needs and risk management requirements for proper documentation. We note, however, that this more flexible standard requires a more rigorous approach to documentation of the actions taken by healthcare personnel. Many staff members are accustomed

to the checkoff system and may fail to adequately document interventions without such a system. Management should consider training and monitoring to ensure compliance with documentation standards. This new method connects the risk assessment to the intervention and allows much more flexibility for staff to deliver timely and effective care rather than basing care on a single number.

Discovery of a Pressure Injury

The initial discovery of a PI is typically documented in the nursing or interdisciplinary progress record section. Any ongoing assessments (and actions, including notification of appropriate medical personnel) should then be documented per facility policy in a wound care form, ADL form, or progress notes but preferably in just one place in the chart for easy access. The response to the discovery of the PI is just as critical as documenting the lesion itself. Documentation should include what immediate interventions were taken, who was notified (the charge nurse/nurse manager, the incoming staff, the physician, the wound care specialist, and/or the family), what topical care was provided to the PI, and any actions that were taken to minimize further pressure/shear (e.g., redistribution of mattress/beds, heel suspension boots, lowering the head of bed, repositioning). Such documentation demonstrates that your facility has a system in place to act quickly and appropriately to changes in the patient's condition.

Correct Identification of the Pressure Injury

The etiology of the PI must be correctly identified in order to provide the most appropriate and effective care. Examine the PI for yourself, review the patient's medical history, make your own judgment, and if it differs with others, discuss your concerns with the team. When in doubt about the etiology or progress of a wound, don't make a speculation in the chart; simply document what you observed. Remember, *objective description beats subjective guessing every time*.

Differentiating between a PI and an ischemic ulcer in the lower extremity can be particularly difficult. For example, is the development of a wound on the lateral aspect of the foot in a person with peripheral vascular disease the result of pressure or arterial insufficiency? The argument could be that "but for" the pressure, the PI would not have occurred. The counterargument could be that "but for" the arterial insufficiency, the tissue could have easily tolerated the pressure exerted on the foot. Objective data are required to solve this dispute. In this case, formal vascular laboratory studies are needed to determine the extent of ischemia and the avoidability of such lesions. Most chronic wounds have distinctive locations, sizes, and presentations and can be differentiated by trained personnel. However, some wounds will defy easy categorization or diagnosis, as many have multiple etiologies. All PI require as much objective data as possible to establish the correct diagnosis and appropriate care plan.

Notification and Participation of the Physician

The patient's primary physician must be notified of any untoward event in a timely manner, including development of a pressure ulcer or other wounds. Good practice requires documentation of when the physician was notified, the response to the notification, any orders given, and the plan for examination and follow-up. Physicians must also meet the standard of care (what a reasonable and prudent provider would do in the same or similar situation) when managing a patient's wound. In facilities with an active wound care team/department, the routine management of the PI is often handed to these specialists by the primary physician. The physician typically signs verbal orders that are written by these specialists. This allows interdisciplinary care and provides maximum potential to heal or mitigate the wound. This does not, however, relieve the physician of the responsibility to monitor the condition of the wound.

Physicians should arrange to examine the wound on a routine basis, have a good understanding of the rationale behind the wound care orders being signed, and be involved in consulting other specialties as needed to maximize healing. Physicians should take the lead in notifying the patient's family about the development of any lesion, just as they would any other negative event that occurs under their care. Pressure injuries are symptoms of underlying medical, physical, and psychosocial problems. They are therefore a multidisciplinary issue involving nursing, nutrition, social work, physical therapy—and medicine—among many other specialties.

Notifying the Patient and Family

Prompt and thorough notification of the patient and family of any new wound or other adverse events is critical to ensure full understanding and participation in care. In some states, such as New Jersey, this is required by law.[12] Full disclosure of all facts related to the development of the wound should be provided: When was it discovered? How was it discovered? What interventions were being taken to prevent the wound? What interventions are being taken now? Give plenty of time for the information to be absorbed, and allow for questions. Many in the lay community believe that pressure injuries or “bedsores” are the result of negligence. An initial negative reaction to an adverse event may be expected, but prompt and full disclosure of the situation will go a long way toward minimizing lingering doubts and suspicions about the adequacy of care in your facility. When discussing the situation with the patient and family, use explanations, not excuses. While the patient’s health status may have played a significant role in the development of the adverse event (e.g., a PI, dehisced surgical incision, or other chronic wound), it is probably best not to dwell on this topic initially as it may be interpreted by family members as “blaming the patient.” Follow-up conversations and briefings with the patient and family may serve as a better time to discuss the realistic goals of healing, once they digest the initial information and the effects of the care plan are better known. When the likelihood is that a wound will not heal, it is also important for the primary care provider to communicate this information along with the rationale supporting this judgment. More in-depth information about communicating with the family regarding pressure injuries can be found elsewhere in the literature.[13]

The patient and family should also be educated that new wounds, especially pressure injuries, are likely to look worse before they look better. A DTI may look rather innocent to the family as a “simple deep bruise” (discoloration) with maybe a little torn skin. The DTI may evolve through a course that can include tissue ischemia, tissue necrosis, necrotic tissue separation, and even ultimate cavitation or ulceration. Lay persons could easily and incorrectly construe such a

change in the wound as substandard care. Preparing the family in advance and setting expectations will reduce the shock of seeing a wound go through this natural process. Any conversations with family members and their response to the information should be promptly documented.

Ongoing Wound Documentation

Wound documentation places a significant burden on the healthcare provider due to the intricate nature of wound assessments. One way to ease this burden is to use logical, well-structured wound documentation forms or computer templates. Check boxes or drop-down lists are recommended for efficient documentation and to limit erroneous entries. Wound assessment forms can be structured in many different ways and will almost always improve the accuracy and consistency of documentation. A glossary of terms should also be developed for use on the wound care form. Drop-down menus, forms, and check boxes, however, are no substitute for narrative nursing assessments when required, and space must be provided in the medical record for such notes as needed.

Frequency of assessment will depend on the wound type, its phase of healing, the resources available to the wound care specialist, and other factors. The International Pressure Ulcer Prevention and Treatment Guideline[14] along with CMS recommend a weekly thorough assessment with daily monitoring of the dressing and wound to assess for complications in long-term care patients.[6] Weekly assessments by a wound specialist allow subtle changes to be noticed that would ordinarily be missed with more frequent inspection. Daily monitoring can be noted in the narrative notes, on treatment sheets, or on a wound assessment form per facility policy but preferably in just one location for ease of reference. Wound documentation should be consistent and concise. Frequent brief, but thorough, notes indicate consistent care.

Wound Photography or Wound Imaging?

Wound photography has become more popular with the advent of inexpensive, quality digital cameras. Two national organizations have position statements about the use of wound photography in wound care.[15,16] What is the rationale behind wound photography? Is it for assessment and diagnosis or just an attempt to mitigate legal liability? Consider *wound imaging* as an assessment and diagnostic tool just like an x-ray or magnetic resonance imaging. If thought of in this manner, wound imaging might be obtained routinely and consistently (per your facility policy and procedure) as with any other assessment and diagnostic imaging. A series of wound images will allow for more efficient and informed interventions and may assist in a legal defense should one become necessary. This regular, methodical approach is in contrast to taking one or two photographs during an inpatient stay to "cover ourselves legally"; taking this approach often backfires. What would any individual, and especially a juror, reacts more positively to a series of detailed photographs showing progress of the wound or one or two photographs taken at odd intervals throughout the patient's stay? One reveals consistency; the other does not.

Wound imaging supplements—but does not replace—the need for written documentation. Each image should have accompanying text discussing what is observed in the photograph. This is similar to obtaining a radiologist's report after medical imaging. Consent for noninvasive medical imaging is rarely required, and the same should also be true for wound imaging as long as the patient cannot be readily identified. Management should clear the consent issue with legal counsel and risk managers (see Chapter 6, Wound Assessment).

Collaboration, Coordination, and Communication

Collaboration, coordination, and communication of all specialty services are essential in maximizing the potential for wound healing. Documentation of “the three Cs” may also demonstrate to the medicolegal expert—and ultimately to a jury—that coordinated, consistent, interdisciplinary care was provided. Most facilities with a wound care team have policies that specify consultation for certain types of lesions. Many wound, ostomy, and continence (WOC) nurses provide both consult services and hands-on care at their facility.[17] If the WOC nurse acts in a consultant role, he or she should examine how consults by other facility services are structured and document in a similar manner. Consultants not only provide recommendations or establish a care plan but also educate other providers on their specialty and the rationale for their recommendations.

Wound care services are also provided by physical therapists in many facilities. As with WOC nurses, consults ideally should be based on the format at their facility and include a care plan and follow-up. Adequate follow-up by either a WOC or staff nurse should be ensured prior to the patient being discharged from physical therapy services. In addition, consults to plastic surgery, vascular surgery, or other surgical specialties are also part of interdisciplinary care. Because there are wide variations in approaches to chronic wound care by these specialties, differences can arise. Therefore, consistent communication and documentation of that communication among the specialties will resolve any differences and is an indicator of quality, interdisciplinary care.

Policies and Procedures: Normative or Positive?

Policies and procedures (P&Ps) establish standards of care within the facility. In any legal proceeding, P&Ps will be scrutinized and compared with the care that is documented in the chart. P&Ps are typically divided into two types of philosophies: normative and positive. A normative P&P describes what care *can* realistically and consistently be provided. A positive P&P describes what care *should* ideally be provided. P&Ps with a positive, ideal focus can cause great trouble in legal proceedings because they set unrealistic and unattainable goals that often exceed a reasonable standard of care.[18]

When establishing P&Ps in your facility, avoid using absolute terms like “will” and “must” and specifying exact time frames for routine nursing interventions unless absolutely necessary. For example, a P&P that states “all patients will have a PI risk assessment every Tuesday and Friday” sets an unrealistic expectation. Missing 1 day or doing the assessment on a Saturday instead of Friday is a violation of your own standard of care. Rewording the P&P to read “all bedfast or chairfast patients should have a PI risk assessment twice a week” gives nursing staff more leeway in their care and documentation. In a lawsuit, “violations” of P&Ps are not always a liability in the defense of such actions. Departures that are explained by and supported by evidence-based/science-based care—and that are fully and completely documented contemporaneously—can often be used to the advantage of the defense. Sometimes, P&Ps must spell out exactly when and where something will or must be done, but mostly they should focus on guiding and educating staff members rather than enforcing strict rules and timelines for care (Box 3-5).

Box 3-5 Preventative Legal Care: Eight Key Areas of Vulnerability for Institutions

1. **Words have meaning—Assessing the legal implications of healthcare facility "policies and procedures"**
   KEY CONCEPT: Healthcare facility policies and procedures are "guidelines," not rules or regulations—and should be created and treated as such. These guidelines should be carefully crafted and periodically reviewed with regard to their clinical currency as well as their legal and healthcare implications. Words such as "never," "must," "shall," and "immediately" should be rigorously avoided.
2. **Assessing compliance with prescribing rules**
   KEY CONCEPT: Healthcare organizations and clinicians should review standing orders to ensure that they are in compliance with prescribing regulations.
3. **Changing and practicing within scope of practice**
   KEY CONCEPT: Healthcare institutions should ensure that caregivers are practicing within their scope of practice with regard to pressure injury assessment and documentation.
4. **Managing expectations and communicating carefully**
   KEY CONCEPT: The people most likely to be asked difficult questions (regarding why, how, and when pressure injuries develop) by patients and their families are not always in the best position to provide an accurate big-picture response. Frontline staff should be trained in how to delegate questions professionally and with compassion.
5. **Clinical documentation**
   a. **Skin assessments**
      KEY CONCEPT: Skin assessments should be conducted regularly and in accordance with the guidelines of a particular institution and in conformance with evidence-based guidelines. Note that the skin assessment is different from the risk assessment and both must be performed.
   b. **Risk assessments**
      KEY CONCEPT: Pressure injury risk assessment guidelines for an organization should be worded in ways that are compatible with federal terminology.

c. **Pressure injury assessment**
KEY CONCEPT: The importance of reasonably complete documentation cannot be overemphasized. Medical record documentation from any provider involved in the care and treatment of the patient may be used to support the determination of whether a condition was present on admission. A “provider” means a physician or any qualified healthcare practitioner who is legally accountable for establishing the patient’s diagnosis.[18]

d. **Charting**
KEY CONCEPT: Good pressure injury documentation should include a wound, periwound and exudate description, measurement and a description of wound care treatments, as well as documentation of pressure redistribution devices and techniques, including support surfaces and turning schedules.

e. **Electronic health records (EHRs)**
KEY CONCEPT: Electronic record systems may not accommodate the documentation needs of pressure injury patients.

f. **Photography**
KEY CONCEPT: Photography has advantages and drawbacks in terms of litigation; know the guidelines set forth by the organization.

g. **Staging**
KEY CONCEPT: Training in the use of NPIAP pressure injury staging is recommended for all healthcare professionals, including physicians. When in doubt about a pressure injury’s stage, all clinicians are encouraged to “describe what they see.” Careful attention should be given to the discharge ulcer assessment.

6. **Preventability—Avoidable, unavoidable, preventable, or never events**
KEY CONCEPT: Government regulations and governmental language can be used to help juries decide healthcare malpractice and wrongful death cases. Understand these

documents and how reimbursement terminology maps onto clinical practice.

7. **Education—The need for learning never ends**
   KEY CONCEPT: Since clinician knowledge of pressure injuries has been linked to pressure injury incidence, initial and ongoing education about best practices is essential. Patient education should do more than address the basics of skin care; it should help patients formulate realistic expectations about their treatment, risks, and recovery.
8. **Preventive clinical care**
   KEY CONCEPT: "Bundles" work and should be implemented when appropriate. While there may be insufficient data for evidence-based product and device selection in pressure injury care, evidence-guided selections can be made.

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PI prevention and treatment[9] practices have undergone significant revisions and changes since the first AHCPR guidelines were released 25 years ago. In addition to these recommendations, guidelines from the NPIAP–EPIAP–PPPIA[14]) and the Wound, Ostomy and Continence Nurses Society (2010) incorporate the latest research and recommendations.[14,17] The guidelines[6,19] are thorough, complete, and easy to read by a wider range of healthcare providers. While developed for long-term care, they are easily adaptable to acute care and other settings and provide comprehensive education and guidance while avoiding "absolute" terminology. Incorporation of these guideline recommendations into your practice indicates that your facility is up-to-date on the latest changes that impact PI care.

Discharge: To Home or Another Facility

The patient and caregiver should have adequate resources to manage the wound upon discharge to the home or another facility.[18] Documentation of this coordination should include teaching strategies and the patient/caregiver response to them, consults with social work or home care, and any equipment or supplies sent home with the patient. Ensure that the patient has adequate follow-up for medical and wound issues.

The discharge assessment should be a thorough and complete “snapshot” of the patient before leaving your facility. As with the admission assessment, you cannot over document in this area. Thoroughly assess and document any wounds and the condition of high-risk PI areas. Document any communication with the receiving facility. Alert the receiving facility to any wounds and describe them in detail. The medical discharge summary typically does not go into significant detail about wound therapy, and thus, the onus for this communication is placed on nursing. List all previous and current wound therapies, which will avoid wasted time in trying therapies that have already yielded little or no results. List all previous and current preventive measures and equipment, such as support surfaces, seat cushions, and heel protectors.

Summary

The patient's chart is an important legal document because it provides a written record of the care provided. It also serves as a means of communication among healthcare professionals about the patient's responses to care. Complete and accurate documentation is not only essential for good patient care but also the basis for mounting a defense in the event of legal action. Make sure entries in the patient's record are legible, thorough, professional, and factual. Ensure that all information is correct, accurate, and timely.

Consistent documentation is a reflection of quality, interdisciplinary care provided to an individual. Chronic wounds are often symptoms of many underlying medical, physical, and psychosocial problems. Documentation of these multiple issues requires a well-structured documentation system. Ensure your documentation system allows healthcare providers to consistently and concisely communicate and access their findings. Policies and procedures should be updated as new research and practices appear. The documentation system should incorporate and reflect these new practices. Such interventions will maximize communication among the interdisciplinary team and help to improve patient outcomes.[18]

PATIENT SCENARIO

Clinical Data

A 66-year-old woman presented herself and was admitted to the emergency department with complaints of headache, nausea, and right upper extremity weakness for 24 hours. Physical examination showed the patient's blood pressure to be 140/86 mm Hg, heart rate 64 bpm, and temperature 100.2°F. Her right extremity grip strength was diminished. Blood test results revealed a decreased leukocyte count and mild hypokalemia.

The patient's prior medical history included a myocardial infarction, hypertension (treated with medication), type 2 diabetes mellitus, and a questionable history of transient ischemic attack.

Before the patient could be sent to radiology for routine x-rays, she suffered precipitous cardiovascular collapse requiring intubation, pressor support, and admission to the ICU. Borderline perfusion was achieved with large doses of pressors (dopamine, norepinephrine) and intravenous fluids. Urine output was diminished but acceptable.

The provisional diagnosis was idiopathic cardiovascular collapse/compromise; sepsis was ruled out.

Hospital Course

The patient remained in the ICU for 24 days. She suffered a cerebral infarct (requiring no surgical intervention) on day 3. Hemodynamic instability lasting 12 days required pressor support and intubation. Total parenteral nutrition begun on day 7.

On day 24, the patient was transferred to a step-down unit with a sacral pressure ulcer that measured 5.6 × 11.2 cm with an average depth of 0.5 cm and significant undermining. Necrotic tissue was evident at the deepest parts of the wound and on some edges.

Surgical, nutritional, and neurologic consults were provided on admission to the step-down unit. A stepwise debridement plan was put into place. The patient subsequently underwent three surgical procedures to close the wound.

The ICU nursing notes 72 hours after admission included the formation of a sacral blister; progress of the wound was documented subsequently. Treatment orders were for saline and other moist dressings. A pressure redistribution bed was not ordered or provided until day 16.

The physician notes clearly indicate that the patient was critically unstable through day 12, maintaining only borderline perfusion. Concern for potential digit loss secondary to tissue hypoxia/coagulopathy/hypotension was noted by the ICU physician and in notes by the vascular consultant until day 12.

Case Discussion

This case was reviewed independently by three law firms, all of whom turned the case down. Expert review uniformly cited the critical, unstable nature of the first 12 days of hospitalization and the critically compromised perfusion as the reason for the PI. The experts also opined that while the PI was clearly a bad result, it was not an unacceptable result of the risk–benefit analysis performed by the caregivers in light of the patient's critically compromised circulatory status. While some experts privately opined that in a perfect world, all sacral pressure injuries would be preventable, in the real world, as in this case, they would not opine that the formation of this injury was the result of medical or other negligence.

From a practice point of view, if a patient's condition is such that the caregivers conclude that measures ordinarily sufficient to delay or prevent tissue breakdown cannot be utilized, clear documentation of the decision-making process and conclusions should be made. In this case, the documentation was adequate in this regard.

Show What You Know

1. **In a medical malpractice trial, what's the role of the jury and the judge?**
   A. Interpreter of the law; finder of fact
   B. Finder of fact; finder of fact and interpreter of the law
   C. Finder of fact; interpreter of the law
   D. Both judge and jury find fact and interpret the law

2. **At trial, how many of the four elements of a medical malpractice claim must a defendant convince a jury that the plaintiff has failed to prove in order to successfully defend against a claim of medical malpractice?**
   A. One
   B. Two
   C. Three
   D. Four

3. **The medical record is:**
   A. a communication tool.
   B. destroyed after 1 year.
   C. a tool to communicate opinions related to a patient's care.
   D. an optional part of health care.

4. **Which one of the following statements about standards of care, practice guidelines, and policies and procedures is *false*?**
   A. They should be reviewed at regular intervals but never amended.
   B. They should be reviewed at regular intervals and amended to reflect new information and research.
   C. They should be based on research and practice experience.
   D. They should be patient outcome oriented and quantifiable.

5. **Given the definition and understanding of deep tissue injury (DTI) from NPIAP, an area of purplish discoloration on an immobile patient's sacrum that was not documented on admission but appears 24 hours later would:**
    A. be classified as an acute medical wound.
    B. be documented as a stage 4 pressure injury.
    C. have occurred prior to admission.
    D. be documented as a stage 3 pressure injury.

6. **Which of the following exemplifies the best way to document frequency of turning a patient in the medical record?**
    A. A 2-hour check box
    B. Regular turning q2 hours
    C. q 4-hour turning
    D. Individualized turning schedule based on patient assessment

7. **Normative policies and procedures describe care that can be:**
    A. realistically and consistently provided.
    B. used for normal staffing situations only.
    C. ideally strived for.
    D. exceeded to achieve magnet status.

Answers to these questions are found on page 681.

References

1. Garner B.A., ed. *Black's Law Dictionary*, 9th ed. New York, NY: West Publishing Company, 2009.
2. Patton, R.M. "Is Diagnosis of Pressure Ulcers Within an RN's Scope of Practice?" American Nurse Today 5(1):20, 2010.
3. National Pressure Ulcer Advisory Panel. National Pressure Ulcer Advisory Panel (NPUAP) Announces a Change in Terminology from Pressure Ulcer to Pressure Injury and Updates the Stages of Pressure Injury [press release]. April 13, 2016. Available at: https:/www.npuap.org/national-pressure-ulcer-advisory-panel-announces-a-change-in-terminology-from-pressure-ulcer-to-pressure-injury-and-updates-the-srtages-of-pressure-injury. Accessed July 7, 19.
4. Sullivan, R. "A Two-Year Retrospective Review of Suspected Deep Tissue Injury Evolution in Adult Acute Care Patients." Ostomy Wound Management 59(9):30-9, 2013.
5. Honaker, J., Brockopp, D., Moe, K. "Suspected Deep Tissue Injury Profile: A Pilot Study." Advances in Skin & Wound Care 27(3):133-40, 2014.
6. CMS State operations Manual: Guidance to Surveyors F 686 Implemented November 28, 2017. Available at http://www.amtwoundcare.com/uploads/2/0/3/7/20373073/som-guidance-to-surveyors-f686-only.pdf. Accessed January 14, 2020.
7. Black, J., Edsberg, L., Baharestani, M., et al. "Pressure Ulcers: Avoidable or Unavoidable? Results of the National Pressure Ulcer Advisory Panel Consensus Conference." Ostomy Wound Management 57(2):24-37, February 2011.
8. Edsberg, L.E., Langemo, D., Baharestani, M.M., et al. "Unavoidable Pressure Injury: State of the Science and Consensus Outcomes." Journal of Wound Ostomy & Continence Nursing 41(4):313-14, 2014.
9. Bergstrom, N., et al. Treatment of Pressure Ulcers. Clinical Practice Guideline. No. 15 AHCPR No. 95-0652. Rockville, MD: Agency for Health Care Policy and Research, December 1994.
10. Defloor, T., Grypdonck, M., De Bacquer, D. "The Effect of Various Combinations of Turning and Pressure Reducing Devices on the Incidence of Pressure Ulcers." International Journal of Nursing Studies 42(1):37-46, 2005.
11. Bergstrom, N., Horn, S., Rapp, M.P., et al. "Turning for Ulcer Reduction: A Multisite Randomized Clinical Trial in Nursing Homes." Journal of American Geriatrics Society 61(10):1705-713, 2013.
12. New Jersey Department of Health and Senior Services. The Department of Health and Senior Services published in the New Jersey Register (40 N.J.R. 1094(a)) final rules implementing the Patient Safety Act (n.J.S.A.26:2H-

12.23to12.25). March 3, 2008. Available at: http://www.njha.com/LibrarySection/pdf/Patient_Safety_Regulations. Accessed May 27, 2014.
13. Fife, C.E., Yankowsky, J.D., Ayello, E.A., et al. “Legal Issues in the Care of Pressure Ulcer Patients: Key Concepts for Healthcare Providers. A Consensus Paper from the International Expert Wound Care Advisory Panel.” Advances in Skin & Wound Care 23(11):493-507, 2010.
14. National Pressure Ulcer Advisory Panel (NPUAP), European Pressure Ulcer Advisory Panel (EPUAP), Pan Pacific Pressure Injury Alliance (PPPIA). Prevention and Treatment of Pressure Ulcers: Clinical Practice Guideline. Perth, Australia: Cambridge Media, 2014.
15. Wound, Ostomy and Continence Nurses Society (WOCN). Professional Practice Series. Photography in Wound Documentation. Available at: http://www.wocn.org/news/93506/Photography-in-Wound-Documentation-Fact-Sheet.htm. Accessed May 27, 2014.
16. American Professional Wound Care Association (APWCA). Resources, Photographic Guidelines. Available at: http://www.apwca.org/Default.aspx?pageId=1491349. Accessed May 27, 2014.
17. Wound, Ostomy and Continence Nurses Society. Guideline for Prevention and Management of Pressure Ulcers. Mount Laurel, NJ: Author, 2010.
18. Brown, G. “Wound Documentation: Managing Risk.” Advances in Skin & Wound Care 19(3):155-65, March 2006.
19. Centers for Medicare & Medicaid Services. Hospital-Acquired Conditions (Present on Admission Indicator). Available at: http://www.cms.gov/Medicare/Medicare-Fee-for-Service-Payment/HospitalAcqCond/index.html. Accessed May 27, 2014.

4

Skin: An Essential Organ

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We'd like to thank Marjana Tomic-Canic, PhD, RN for her contributions to the previous editions.

Objectives

After completing this chapter, you'll be able to:

- discuss the functions and different layers of the skin
- identify the components of a skin assessment
- evaluate skin changes associated with the aging process
- differentiate between skin assessment and wound assessment.

Skin Anatomy and Physiology

The skin is a multifunctional and complex organ that provides the human with critical physiological and homeostatic mechanisms that ensure survival. Not only is it the largest organ of the body, but it is a barrier between the external environment and the internal organs. “It protects against trauma from water, chemicals, microorganisms, mechanical stress and radiation.”[1] Aside from protection, the skin provides water balance, temperature regulation, sensation, and hormonal functions such as synthesis of vitamin D and sex hormone metabolism. Human skin is composed of two distinct layers: the epidermis, the outermost layer; and the dermis, the second layer (Fig. 4-1). The dermal–epidermal junction, commonly referred to as the basement membrane zone (BMZ), separates the two layers. Under the dermis lies a layer of loose connective tissue, called subcutaneous tissue or hypodermis (Table 4-1).

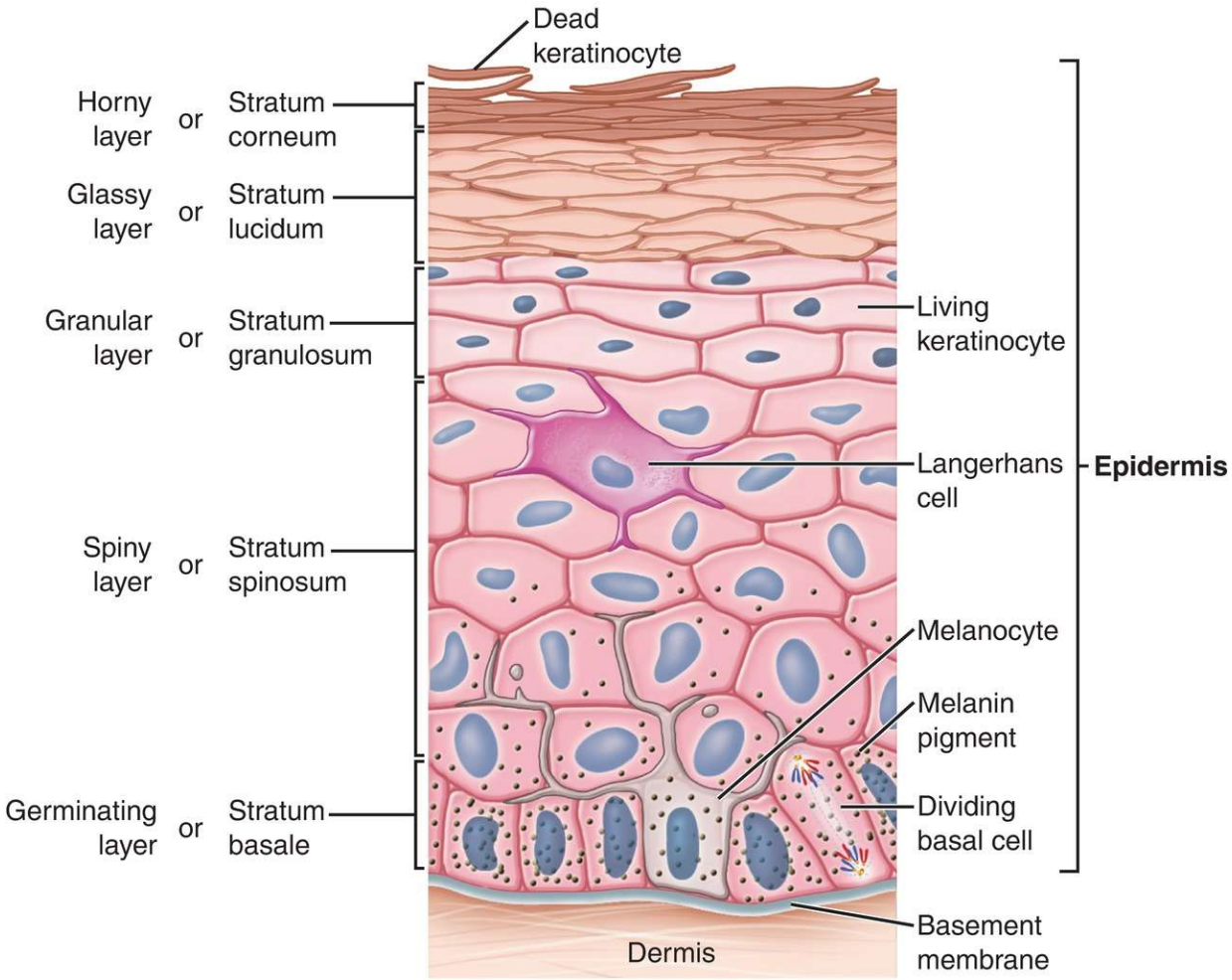

Figure 4-1. Layers of the skin. Two distinct layers of skin, the epidermis and dermis, lie above a layer of subcutaneous fatty tissue (also called the hypodermis). The dermal–epidermal junction (also called the basement membrane zone) lies between the dermis and epidermis.

Table 4-1 Skin Layer Functions

Skin Layer	Characteristics	General Function
This table shows characteristics and general functions of each layer of the skin.		
Epidermis	• Outer layer of skin • Consists of five layers (or strata): corneum, lucidum, granulosum, spinosum, and basale (or germinativum) • Repairs and regenerates itself every 28 days	• Protective barrier (sun damage, transepidermal water loss [TEWL]) • Organization of cell content • Synthesis of vitamin D and cytokines • Division and mobilization of cells • Maintaining contact with dermis • Pigmentation (contains melanocytes) • Allergen recognition (contains Langerhans cells) • Differentiates into hair, nails, sweat glands, and sebaceous glands
Dermis	• Consists of two layers—papillary dermis and reticular dermis—composed of collagen, reticulum, and elastin fibers • Contains a network of nerve endings, blood vessels, lymphatics, capillaries, sweat and sebaceous glands, and hair follicles	• Supports structure • Mechanical strength • Supplies nutrition • Resists shearing forces • Inflammatory response
Subcutaneous tissue (hypodermis)	• Composed of adipose and connective tissue • Contains major blood vessels, nerves, and lymphatic vessels	• Attaches to underlying structure • Thermal insulation • Storage of calories (energy) • Controls body shape • Mechanical "shock absorber"

Embryology is the study of organismal development from fertilization to birth and is an often overlooked topic that can lend insights into structure and function. The complex functions of the skin as an organ are reflected by its complex development in utero. As a system with multiple specialized structures, it has contributions from all embryonic layers. As the single-celled fertilized ovum divides and multiplies, several layers develop including ectoderm and mesoderm. Ectoderm evolves into stratified epithelium, while mesoderm differentiates into connective tissue that includes sensory receptors, nerve endings, fibroblasts, and blood vessels. Embryonic ectoderm contributes to epidermal appendages including hair follicles and sebaceous and sweat glands, while connective tissue differentiates into dermis and subcutaneous tissue. Another embryonic structure called the neural crest develops into melanoblasts that are responsible for pigment production resulting in skin color.[2]

The epidermis is a thin, avascular layer that regenerates itself every 4 to 6 weeks. It's divided into four layers or strata (presented in order from the outermost layer inward).[1]

- *Stratum corneum (horny layer)*—consists of dead keratinocyte cells; the scale flakes and sheds; is easily removed during bathing activities and more traumatically by scrubbing the surface of the skin
- *Stratum granulosum (granular layer)*—also contains Langerhans cells in addition to keratinocytes
- *Stratum spinosum (spiny layer)*—contains keratinocytes and Langerhans cells
- *Stratum basale (germinating layer)*—single layer of epidermal cells (keratinocytes); contains melanocytes; can regenerate

A fifth layer, the *stratum lucidum (glassy layer)*, lies between the stratum corneum and the stratum granulosum. This packed translucent line of cells is found only on the palms and soles and not seen in thin skin.

The epidermis is composed of keratinocyte cells. Basal keratinocytes (stratum basale) have the capacity to divide with half of the cells, giving rise to suprabasalar layers of epidermis and the other half staying in the basal layer ready for another cell cycle. Once basal keratinocytes leave the stratum basale, they start the process of differentiation, during which they die. This process involves making insoluble proteins and their cross-link, which, along with lipids and membrane components, form the insoluble, horny stratum corneum layer.[2–7] In order to maintain the barrier, keratinocytes have the capacity to completely regenerate the epidermis. If damaged (such as wounded, burned, exposed to ultraviolet [UV] light or chemicals), keratinocytes, in order to repair the damage, change their biology. Instead of differentiating, they become “activated” and start to divide rapidly, and, in the case of a wound, they migrate over the gap to repair the damage.[5] They also signal to other neighboring cell types, such as fibroblasts, Langerhans cells, and melanocytes, that the skin barrier is compromised and that they’re needed to help repair the damage. Once the damage is repaired, the keratinocytes cease their activation and resume their normal differentiation process.[7–9]

The BMZ divides the epidermis from the dermis. It contains fibronectin (an adhesive glycoprotein), type IV collagen (a non–fiber-

forming collagen), heparin sulfate proteoglycan, and glycosaminoglycan.[10] The BMZ has an irregular surface—called rete ridges or pegs—projecting downward from the epidermis that interlocks with the upward projections of the dermis. The part of the dermis beside the rete pegs is called the papillary dermis. The two interlocking sides resemble the two sides of a waffle iron coming together. This structure anchors the epidermis to the dermis, preventing it from sliding back and forth. These underdeveloped structures are absent in premature neonates but develop after birth.[1] As skin ages, the basement membrane flattens, and the area of contact between the epidermis and dermis is decreased by 50%, thus increasing the risk of skin injury by traumatic, accidental separation of the epidermis from the dermis (Fig. 4-2). The deeper dermis below the rete pegs is named the reticular dermis, and this provides support for the papillary dermis above and the subcutaneous fat below.

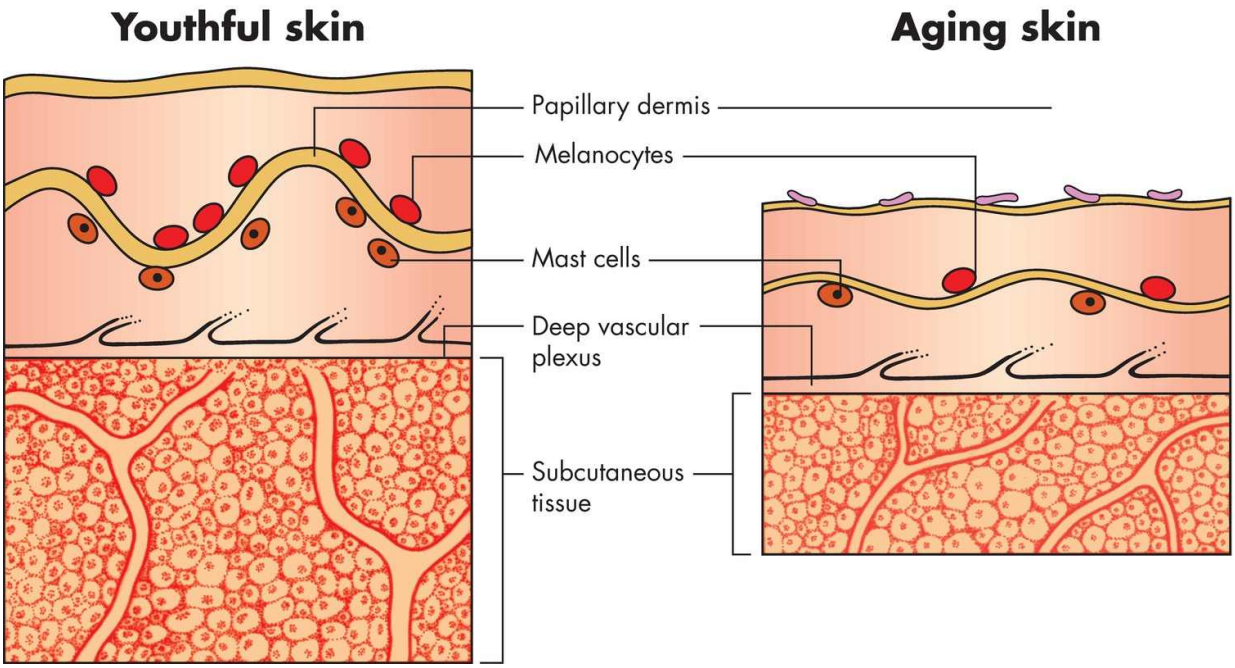

Figure 4-2. Effects of aging on the basement membrane zone (BMZ). The illustrations below show the effects of aging on the BMZ. Specifically, the basement membrane flattens, reducing the area of

contact between the epidermis and the dermis by 50%.

The dermis is an essential part of the skin and is commonly referred to as the "true skin."[9] As the second layer, it's the thickest layer and is composed of many cells. The major proteins found in this layer are collagen and elastin, which are synthesized and secreted by fibroblasts; collagen forms up to 30% of the volume or 70% of the dry weight of the dermis.[10–12] The dermis is a matrix that serves to support the epidermis. The composition of the two dermal layers is different. It's divided into two areas, the papillary dermis and the reticular dermis.[11]

- The *papillary dermis* is composed of collagen and reticular fibers. Its distinct, unique pattern allows fingerprint identification for each individual. It contains capillaries for skin nourishment and pain touch receptors (pacinian corpuscles and Meissner's corpuscles).
- The *reticular dermis* is composed of collagen bundles that anchor the skin to the subcutaneous tissue. Sweat glands, hair follicles, nerves, and blood vessels can be found in this layer.

The main function of the dermis is to provide tensile strength, support, and moisture retention and deliver nutrients, blood, and oxygen to the skin.[11] It protects the underlying muscles, bones, and internal organs. The dermis also contains the sebaceous glands that secrete sebum, a substance rich in oil that lubricates the skin surface preventing the stratum corneum from dehydration and a loss of barrier function. Furthermore, it also contains hair follicles that are the source of multipotent stem cells that have the capacity to restore a wounded epidermis.[8]

The subcutaneous fat tissue, or hypodermis, attaches the dermis to underlying structures. Its function is to promote an ongoing blood supply (blood reservoir) to the dermis for regeneration. It's primarily composed of adipose tissue, which provides a cushion between skin

layers, muscles, and bones. It promotes skin mobility, molds body contours, and insulates the body.

Skin Roles and Functions

Skin is an important organ whose diverse functions are not always appreciated (Table 4-2). In adults, the skin weighs between 6 and 8 pounds (2.7 to 3.6 kg) and covers over 20 ft^2 (1.9 m^2). Skin thickness varies from 0.5 to 6 mm according to its location on the body; for example, skin can be as thin as 1/50 inches on the eyelids and as thick as 1/3 inches on the palms and soles, where greater protection is needed. The skin receives one-third of the body's circulating blood volume—an oversupply of blood compared to its metabolic needs.

Table 4-2 Skin Roles and Functions

- Protection
  - Acid mantle suppresses bacteria growth
  - Moisture barrier/water resistance
- Immunity
- Thermoregulation
  - Excretion of sweat regulator
- Sensation
  - Pleasure
  - Pain
- Sweat
- Storage and synthesis
  - Excretions/absorption/synthesis vitamin D
  - Storage lipid, H_2O
  - Blood reservoir
- Water barrier

From Damjanov, I. *Pathology for the Health-Related Professions*. Philadelphia, PA: W.B. Saunders, 2000.

The normal range of skin pH is 4 to 6.5 in healthy individuals.[13,14] This “acid mantle” helps to maintain a normal skin flora by serving as a protective barrier against bacterial and fungal infections (Fig. 4-3). It also supports the formation and maturation of epidermal lipids and assists in maintaining their protective barrier function. The acid mantle also provides indirect protection against invasion by microorganisms and protection against alkaline substances.[13] If the acid mantle loses its acidity, the skin becomes more prone to damage and infection. Frequent use of soap products especially antibacterial soaps with a high alkaline pH and overwashing can remove most of the stratum corneum and its ability to serve as a protective barrier. Alternatively, several skin conditions can *increase* the skin’s surface pH, including chronic wounds, eczema, contact dermatitis, atopic dermatitis, and dry skin.[15,16] Systemic diseases may also increase the skin’s surface pH, for example, diabetes, chronic renal failure, and cerebrovascular disease.[17]

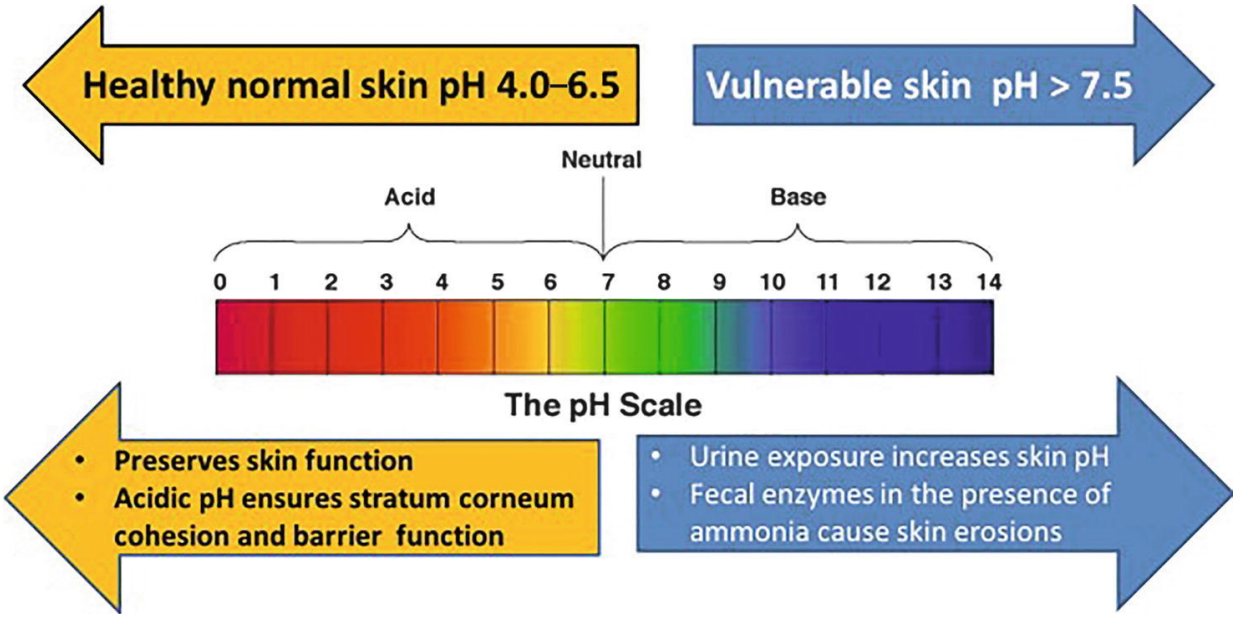

Figure 4-3. Skin and pH. (Source McNichol, L.L., Ayello, E.A., Phearman, L.A., et al. Incontinence-Associated Dermatitis: State of the Science and Knowledge Translation. *Advances in Skin & Wound*

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The skin's major functions are protection, immunity, thermoregulation, sensation, sweat, storage, synthesis, and endocrine[12] and water barrier.[18,19] Skin protects the body by serving as a barrier from invasion by bacterial organisms. The epidermis synthesizes natural antimicrobials called defensins.[20] Because staphylococcal species (such as *Staphylococcus aureus* or *Staphylococcus epidermis*) tolerate salt, they may be present in large numbers as resident bacteria on the skin.[17,19] Another organism found on the skin is yeast, which is commonly found on the trunk and ears, and dermatophyte fungus has a susceptibility to invade moist spaces between the toes.[17,20] In addition, skin makes its own antimicrobial peptides called defensins.[21–23]

The skin is involved in hormone synthesis and metabolism, including sex steroids. In males, DHEA and androstenedione are converted to testosterone or 5α-DHT in the skin. The skin has also been identified as an important extragonadal site of estrogen synthesis, specifically the compound known as 17β-estradiol (estradiol).[24]

Sensation is a key function of the skin. Areas that are most sensitive to touch have a greater number of nerve endings.[21] These include the lips, nipples, and fingertips. In humans, the fingertips are the most sensitive touch organ and enable us to correctly identify objects by touch (stereognosis) rather than by sight. Many tactile corpuscles lie at the base of hair follicles, and shaving reduces the tactile sensibility of that skin area. In hairless body regions, the tactile corpuscles are called Meissner's corpuscles.[21] Pleasurable, firm touching sensations, such as from a massage or hugs of affection, are transmitted via the skin as they generate nerve transmissions through these tactile corpuscles.

Itch is one of the alarm sensations of the skin and serves as a defense mechanism. Chemicals that are released after skin injury

may promote the inflammatory process and can also induce itch or pain. Itch and pain are in close regulatory relationship—for example, central pain inhibition may enhance the act of itching while also inhibiting the act of itching.[24,25]

Somatic pain (from the outer body surfaces and framework) is also communicated through the skin. Superficial (acute) pain to a local area is usually transmitted by very rapid nerve impulses by A-delta fibers[24] and tends to be sharp but ceases when the pain stimulus stops. Deep (chronic) pain impulses are transmitted slowly over the smaller, thinly myelinated C fibers. In contrast, this type of pain tends to spread over a more diffuse area, lasts for longer periods of time, and remains even after the pain stimulus is gone.[24] As a sign of possible skin injury, pressure also serves as a protective warning sensation.

Temperature regulation and fluid and electrolyte balance are achieved in part by the skin. Thermoregulation is controlled by the hypothalamus in response to internal core body temperature. Peripheral temperature receptors in the skin assist in this process called *temperature homeostasis.*[26] By losing a copious amount of water—for example, sweating—through the skin, lungs, and buccal mucosa, homeostasis of body temperature is maintained. Skin temperature is controlled by the dilatation or constriction of skin blood vessels. When body core temperature rises, the body will attempt to reduce its temperature by releasing heat from the skin. This is accomplished by sending a chemical signal to increase blood flow in the skin from vasodilation, thus increasing skin temperature.

Practice Point

Increased temperature → Skin blood vessel vasodilation → Heat loss from epidermis → Body maintains temperature homeostasis.

In contrast, the opposite occurs when the body's core temperature is reduced; the chemical signal causes decreased blood flow from vasoconstriction, thus lowering the skin's temperature.[26,27]

Practice Point

Decreased temperature → Skin blood vessel vasoconstriction → Heat conservation → Body maintains temperature homeostasis.

The skin also aids in the excretion of end products of cell metabolism and prevents excessive loss of fluid. Other important functions of the skin include its manufacturing ability and immune functions.[28] For example, when exposed to UV light, the skin can synthesize vitamin D that becomes active through metabolism in the liver and kidney to the 1.25 OH vitamin D.[29] Although the skin's hypersensitivity responses in allergic reactions are commonly seen, the skin's role in immune function isn't always fully appreciated. Indeed, Langerhans cells and tissue macrophages, which play an important role in digesting bacteria, as well as mast cells, which are needed to provide proper immune system functioning, are all present in the skin.[3,10,18] In addition, keratinocytes are very powerful in generating rapid inflammatory response because they contain prestored, proinflammatory signals, such as interleukin-1, which gets released the moment the barrier is broken and is considered the first signal of wounding.[30] We are more likely to cause an allergic reaction by exposing the skin to potential allergens than through oral ingestion of a potential allergen that is often metabolized or broken down to smaller molecules in the gut.

Aging and the Skin

Intrinsic and Extrinsic Causes of Aging Skin

Changes that occur to human skin as it ages are classified as intrinsic and extrinsic. Intrinsic refers to physiologic changes taking place in the aging process and are most evident in sun-protected areas, while extrinsic refers to environmental influences. It is sometimes difficult to separate intrinsic from extrinsic factors due to the pervasive impact of diet and lifestyle factors, but there are profound genetic and ethnic differences in the body's response to both.

The photo in Figure 4-4 presents a comparison between a 22-year-old female medical resident and a 91-year-old female, illustrating thinning of the skin with obvious visualization of deeper anatomic structures.

Figure 4-4. This photo presents a comparison between a 22-year-old female medical resident and a 91-year-old female, illustrating thinning of the skin with obvious visualization of deeper anatomic structures. (Photo courtesy of J. Levine.)

Another major factor impacting both intrinsic and extrinsic aging is oxidative stress, leading to macromolecular damage and cell senescence. Changes of aging depend largely upon homeostasis between free radical production and the proper working of repair systems. The term reactive oxygen species (ROS) refers to free radical and nonradicals that contain an oxygen atom. ROS are by-products of cell respiration that takes place in mitochondria during oxidative phosphorylation but are generated by other cellular structures such as peroxisomes and endoplasmic reticulum.[31] Not only does ROS increase with age, but aging is accompanied by reduced antioxidant activity and decreased DNA repair capability.

Extrinsic causes of aging skin are also mediated by oxidative stress and ROS. The most important cause of aging skin is UV light from the sun, which is divided into UVA and UVB. UVA is considered more damaging to photoaging because of its deeper penetration into the dermis. Other environmental factors recognized as extrinsic causes of aging include cigarette smoke, ozone, and airborne particulate matter with adsorbed polycyclic aromatic hydrocarbons (pollution in the air).[32] These extrinsic factors generate free radicals and ROS that overwhelm the body's natural antioxidant defenses and stimulate lipid peroxidation reaction cascade, which in turn releases proinflammatory mediators that include matrix metalloproteases (MMPs). Mitochondrial DNA mutations resulting from ROS lead to defective electron transfer activity and oxidative phosphorylation.

Telomere shortening is a known result of oxidative insults resulting from environmental factors but is also associated with psychological stress.[33] Telomeres are repetitive DNA sequences at the end of linear

DNA that shorten each time a cell divides and ultimately leads to cessation of cell division and apoptosis or programmed cell death.

Polycyclic aromatic hydrocarbons adsorbed to airborne particulate matter induce xenobiotic metabolism that releases ROS and MMPs that accelerate aging. Xenobiotic metabolism refers to the metabolic pathways the body uses to eliminate environmental toxins such as polycyclic aromatic hydrocarbons that are adsorbed to airborne particulate matter.[32]

Changes in Aging Skin

Age-related changes in the dermis are numerous, and their cumulative effect increases susceptibility to environment and internal stresses that lead to increased fragility and impaired wound healing. The most striking is the approximately 20% loss in dermal thickness that accounts for the paper-thin appearance of aging skin.[34,35] This decrease in dermal cells and proportional reduction in collagen fibers, blood vessels, nerve endings, and collagen lead to altered or reduced sensation, thermoregulation, rigidity, moisture retention, and sagging skin (see Fig. 4-4).[34,36]

A decrease in differentiation and formation of the stratum corneum is also detected in aging skin. In the epidermis, there is reduced keratinocyte proliferation and turnover time, and surface pH is less acidic. Desquamation is less effective, and lipid biosynthesis in the stratum corneum is impaired. There are decreased melanocytes that reduce the pigment response to protect the skin from UV radiation and decreased Langerhans cells that process microbial antigens and present them to other immune system cells. This is accompanied by altered T- and B-cell function and a general proinflammatory environment that is now an accepted component of the aging process.

The dermal–epidermal junction is flattened with smoothing of the rete ridges and decreased adhesion of this critical mechanical defense barrier. The dermis becomes atrophic with reduced numbers of fibroblasts and mast cells, and collagen becomes disorganized with change in synthesis from type I to type III. There is decreased synthesis of elastin, and elastic tissue is degraded with overall loss of elasticity.[37] Flattening of the dermal–epidermal junction is a principal cause of fragility that leads to skin tears.

Reduced collagen deposition in elderly skin could explain the development of dermal atrophy and might relate to poor wound healing.[37] The subcutaneous fat below the dermis consists primarily of adipose tissue and provides mechanical protection and insulation. Its loss during aging results in parallel reductions in these protective

functions. Subcutaneous tissue undergoes site-specific atrophy in such areas as the face, dorsal aspect of the hands, shins, and plantar aspects of the foot, increasing the energy absorbed by the skin when trauma occurs to these areas.[37]

Many of the changes in aged skin are linked to the hormones estrogen and androgen. Decreased estrogen in menopausal women, similar to ovariectomized mice, leads to a decrease in collagen deposition, slower epithelialization, and delayed wound healing. These effects may be reversible by hormone replacement therapy (HRT).[38] In addition, a genetic polymorphism in estrogen receptor-beta has been linked to a predisposition to venous ulcerations in both male and female patients.[35] In contrast to estrogen, which is beneficial for wound healing, androgens are implicated in the etiology of venous ulcerations.[39] In addition, use of inhibitor (antagonist of androgen) or castration in mice leads to increased collagen deposition and acceleration of wound healing.[40]

Decreased mechanoreceptors including Meissner's and Pacini's corpuscles result in diminished sensation to light touch, pressure, and vibration. A decrease in pain perception may make elderly people more vulnerable to traumatic environmental insults such as wearing tight shoes, stepping on an object, or hitting legs on the side of a chair. Aging skin is also less able to manufacture vitamin D when exposed to sunlight.[34,35] The number of Langerhans cells and mast cells diminishes in aging skin, translating into decreased immune function.[34–41] Medications also have adverse effects on the skin's immune function. For example, steroids cause thinning of the epidermis.[34,41,42]

There are decreased pilosebaceous units that are composed of hairs, sebaceous glands, and arrector pili muscles, which contribute to decreased sebum production.[42] Impaired thermoregulation results from loss of subcutaneous fat and results in decreased energy stores along with decreased autonomic nerves from the sympathetic nervous system and decreased dermal vascularity. Loss of sweat glands also contributes to impaired thermoregulation as well as decreased ability to manage water balance in response to antidiuretic hormone.

Photoaging Versus Natural Aging

Skin changes seen with aging are accelerated by sun exposure, specifically due to UV radiation.[34,41] UV irradiation causes local inflammation and local immunosuppression with DNA damage. Skin damage comes from UVA and UVB, although most burning and carcinogenic waves come from UVB with potentiation from UVA. Changes induced by photoaging are superficially similar but differ slightly under the microscope. Photodamaged skin looks coarse, rough, and wrinkled and is prone to developing malignancy. The UVA waves penetrate deeper in the skin, can travel through the car window, and cause wrinkling and benign brown pigment on the surface of the skin most commonly seen on the face and dorsum of the hands. UVA light can add to the UVB carcinogenesis, but UVB is the predominant wave length associated with skin cancer.[43,44]

For sun protection, a sunscreen with UVB protection as measured with the sun protection factor (SPF) should be applied prior to any other cream or ointment. The SPF only measures UVB protection. An SPF of 30 to 60 is adequate but may need to be reapplied every 4 hours or after water exposure or sweating. For UVA protection, check the label for broad spectrum of action or UVA/UVB coverage.[43] Individuals may develop an allergy or photo allergy to sunscreen chemicals. Sensitive individuals may be best treated with a physical sunscreen (physical blocker) containing micronized zinc oxide or titanium dioxide. In addition to a sunscreen, a hat with a 3-inch brim, protective clothing, and limiting outdoor sun exposure between 11-3 can prevent excessive damage.[43,44]

Ultraviolet light can cause premalignant and malignant skin changes. An actinic keratosis is a scaly red papule or plaque usually less than 1 cm on sun-exposed areas of the face and arms especially the dorsum of the hands. These lesions can evolve into Bowen's disease (often slightly larger lesions that may be associated with a cutaneous horn—thick protruding keratin) on a red base, and they can even evolve into a squamous cell carcinoma (keratotic papules, often with a red base and maybe painful). A basal cell carcinoma is even more common and can have a translucent pearly papule with surface

blood vessels (telangiectasias) on the surface. These lesions bleed easily and can often have a central ulcer.[44,45]

Each blistering burn doubles the incidence of skin cancer lifelong, including melanoma. If you want to see how much photoaging versus natural aging you have, perform the two-cheek test next time you have a bath! With a handheld mirror and a full-length mirror behind you, look at the amount of pigment, wrinkles, and small blood vessels on the face, and compare this to the buttocks that, for most of us, only have natural aging!

Practice Point

A melanoma is the most serious of the skin cancers and often is described with the mnemonic ABCD[46]:

- **A**symmetry—one side doesn't look like the other
- **B**order—irregular like a ski slope
- **C**olor variability—black, too much melanin; blue, the melanin is deep by the effect of bending light in denser tissue; white, regression; and red, inflammation
- **D**iameter—anything greater than 6 mm—an eraser on the end of a pencil

Aging skin is less able to retain moisture due to a decrease in dermal proteins, which leads to oncotic pressure shifts and diminished fluid homeostasis, thereby putting elderly people at risk for dehydration. Normal water content of the skin is 10% to 15%. Below 10%, the skin becomes dry and is more vulnerable to damage.[35] Because soap increases the skin's pH to an alkaline level, using emollient soap and bathing every other day, instead of every day, can decrease the incidence of skin injury, such as skin tears, in elderly patients.[13]

An elderly person's skin is less stretchable due to a decrease in elastin fibers.[34,37,41] Because of the thinning of the epidermal layer,

the skin becomes a less-effective barrier against water loss, bruising, and infection, accompanied by impaired thermal regulation, decreased tactile sensitivity, and decreased pain perception.[34,35,44,45] Due to a decreased amount of dermal proteins, the blood vessels become thinner and more fragile, thereby leading to a type of hemorrhaging known as senile purpura (see Fig. 4-5). Hematomas can dissect into surrounding skin, usually over the extensor surfaces of the hands and forearms, resolving to leave brownish discoloration caused by hemosiderin and melanin deposits. The appearance of dissecting hematomas can mimic dermal changes associated with bleeding diathesis or impaired clotting system. These hematomas are more common on sun-damaged skin where the photodamage and chronological aging are more prominent on the dorsum of the hands or extensor surfaces of the arms. Adding also to the risk of hematoma formation is the common use of anticoagulants in aging persons including traditional agents such as warfarin and newer agents such as apixaban and rivaroxaban.

Figure 4-5. Senile purpura with xerosis.

Skin Assessment

A skin assessment captures the patient's general physical condition, based on careful inspection and palpation of the skin. Be sure to start with a thorough history of the patient's skin condition, including any allergies to skin products, medications, topical skin and wound products, and food. A skin assessment should include an actual observation of the entire body. It differs from a wound assessment in that a skin assessment looks at the patient's entire body and not just open wounds. A review of the patients' history should capture the usual skin care practices including bathing routine (shower, or bath) and type of skin/soap products used or preferred.

A through skin assessment includes inspecting all areas of the skin, from head to toe. Always check the nails, scalp, hair, and mucous membranes as well.[47,48] A minimal skin assessment should include the following five parameters: temperature, color, moisture, turgor, and skin integrity. Lacking consensus in the literature as to what constitutes a minimal skin assessment, the authors recommend these five parameters for skin assessment (Table 4-3). All examinations begin with general inspection of the skin, followed by palpation, describing what you see and documentation.

Table 4-3 Elements of a Skin Assessment

At a minimum, a skin assessment should include the following:

Temperature

- Normally warm to the touch
- Warmer than normal could signal inflammation
- Cooler than normal could signal poor vascularization (Compare bilateral extremities for temperature changes)

Color

- Culturally component skin assessment: awareness of differences across skin pigmentation
- Normal color tones: light ivory to medium, to deep brown, black, yellow to olive, or light pink to dark, ruddy pink
- Intensity: paleness may be an indicator of poor circulation or anemia
- Erythema, varies with natural skin color (Sommers)
- Hyperpigmentation or hypopigmentation reflect variations in melanin deposits or blood flow

Moisture

- Dry (xerosis)
- Moisture-associated skin damage (incontinence, perspiration periwound maceration, peristomal)
- Edema, mild, moderate, severe

Turgor

- Tented skin normally returns to its original state quickly
- Slow return to its original shape (dehydration or effect of aging)

Integrity

- No open areas
- Type of skin injury (use the appropriate classification system to identify and record injury type)

Patient condition, clinical resources, and facility practices may support a more comprehensive and involved skin assessment. Usually comprehensive assessments are completed by those clinicians specializing in skin and dermatology. Comprehensive assessments would include skin inspection, palpation and olfaction, and observation of hair and nails and finally any skin alterations seen (see Table 4-4).

Table 4-4 Elements of a Comprehensive Skin Assessment

Consider the following criteria when performing a comprehensive skin assessment

Inspection

- Normally smooth, slightly moist, and same general tone throughout
- Tone depends on the patient's melanocytes—skin pigmentation continuum can vary from light ivory, deep brown, black, yellow to olive, light pink to dark ruddy pink, or red
- Pigmentation can exhibit:
  - Pallor: mucosa, conjunctivae
  - Cyanosis: nail beds, conjunctivae, oral mucosa
  - Jaundice: sclerae, palate, palms
  - Hyperpigmentation: increased (results from variation in melanin deposits or blood flow; palpate for skin temperature and for edema over these areas to assess circulation)
  - Hypopigmentation: decreased vascular/venous patterns, usually symmetric
  - Scars and bruises for location, color, length, and width

Palpation

- Moisture: perspiration
- Edema: extremities, sacrum, eyes
- Tenderness
- Turgor, elasticity
- Texture

Olfaction

- Normal body odor
- Absence of pungent odor
- May indicate presence of bacteria or infection
- Poor hygiene

Observation of Hair and Nails

- Hair
  - Hirsutism: excessive body hair
  - Alopecia: hair loss
- Nails (can reflect the patient's overall health)
  - Color, shape, contour
  - Clubbing, texture, thickness

Skin Alterations

- Previous scars
- Graft sites
- Healed ulcer sites

Skin Assessment Parameters

Skin Temperature

Skin temperature should be assessed by comparing bilateral extremities for coolness or warmth using the back of the hand.[47] Warmth can be an indication of infection or inflammation especially of the deeper structures, while coolness could signal diminished vascular supply. Some clinicians use noncontact infrared thermometers to assess skin temperature.

Skin Color

Skin color can reflect various skin conditions. Assessing for changes in a person's normal skin tone can be extremely challenging. In order to evaluate skin color, one needs adequate light (natural light or halogen is preferred) to see if there are any changes in the skin tone. Healthcare professionals are caring for many diverse populations with varying skin colors/tones.[19] Clinicians need to keep this in mind, when assessing the nuances of changes in patients' skin.[19,48,49] Differences in skin color pigmentation can be described using Fitzpatrick's skin categories.[50] The four categories were expanded to include brown and black skin types.[19] Each of the six categories includes the skin's response to UV radiation[19] (Table 4-5). Any trauma or injury to the skin can produce postinflammatory hypo- or hyperpigmentation when injury disrupts the dermal–epidermal junction where most of the melanocytes reside. This can also occur with specific skin disease including the end stage of eczema or dermatitis, lichen planus, or collagen vascular diseases such as lupus erythematosus.[19] Jaundice can indicate biliary tract disease or liver problems; pale yellow skin can indicate a renal problem. A red or flushed face can indicate excessive ETOH intake, fever, or localized inflammation.

Table 4-5 Variation of Skin Colors

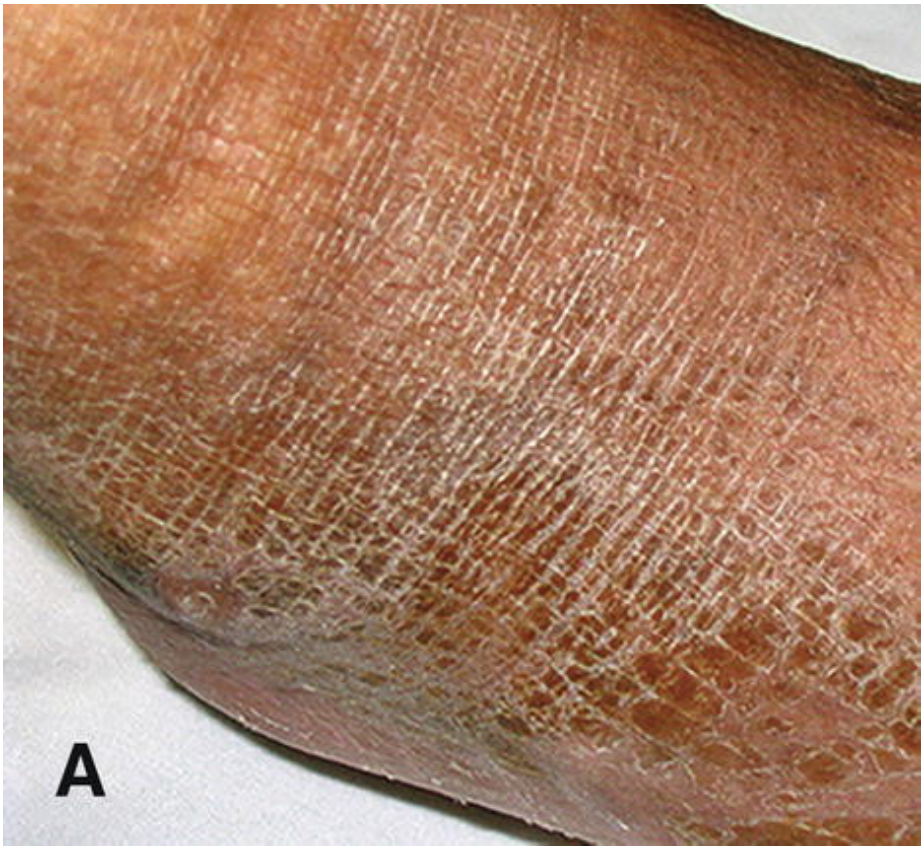
Skin Color	Description	Burns	Tans
1. Pale white	Red/blond hair Blue eyes, freckles	Always	Never
2. Fair white	Red/blond hair Blue, green, hazel eyes	Usually	Minimally
3. Cream white	Any hair color Any eye color	Sometimes (mild)	Uniform
4. Moderate brown	Mediterranean skin	Rarely	Always
5. Dark brown/yellow	Mid-East Asian	Very rarely	Easily
6. Deeply pigmented	Dark brown/black skin	Almost never	Very easily

The first three skin types are most likely to have chronic sun damage. Lighter skin with the excessive sun-related changes increases the likelihood of an individual developing skin cancer.

From Ayello, E.A., Sibbald, R.G., Quiambao P.C.H., et al. “Introducing a Moisture-Associated Skin Assessment Photo Guide for Brown Pigmented Skin.” *World Council of Enterostomal Therapists Journal* 34(2):18-25, 2014; Skin Inc. The Fitzpatrick Skin Type Classification Scale. November 2007. Available at: http://www.skininc.com/skinscience/physiology/10764816.html. Last accessed June 2, 2014.

Moisture Balance

Assess skin for the level of moisture seen externally or felt on the surface. The stratum corneum requires a 10% moisture content to stay intact.[15] Too little moisture results in dry, scaly, flaky skin commonly referred to as xerosis (Fig. 4-6). Dry skin can be caused by alkaline soaps, overbathing, and hypothyroidism. The elderly population normally have dry skin/xerosis. Too much moisture on the skin surface can result in moisture-associated skin damage (MASD). MASD will be discussed in detail in Chapter 13.



B

C

Figure 4-6. Xerosis terminology. **(A) Mild.** Dry skin with minimal flaking. Treatment: Hydrate the skin using a moisturizing agent frequently. **(B) Moderate.** Dry skin with a scaly, fish-like appearance that's easily rubbed off the skin surface. Treatment: Use an exfoliating emollient moisturizing agent. **(C) Severe.** Cracking, parched appearance of skin that resembles dry earth. Treatment: Use moisturizer with urea, AHA, or lactic acid to exfoliate calloused dry skin.

Xerosis

Xerosis is the medical term for dry skin.[51] In xerosis, the skin appears dry, scaly, and flaky.[51] Older adults are prone to xerosis due to loss of sebaceous and sweat glands. Although there is a xerosis scale, it's not widely used in clinical practice. Clinicians generally classify xerosis as mild, moderate, and severe. The term xerosis has no particular diagnostic implication. Xerosis can be caused by environmental factors or a symptom of an underlying disease. For this reason, a patient's complaint of "dry" skin needs to be explored further. Skin exposure to a dry environment, such as central heating, wind, temperature extremes, or air conditioning, can all lead to xerosis.

The goal in treating xerosis is to protect the skin from excessive transepidermal water loss (TEWL) and return the natural moisturizing factors to the stratum corneum. This is best accomplished by using moisturizing agents that contain lipids—an essential component in forming an impervious barrier, or seal, on the stratum corneum, thus preventing further water loss. As water is retained, the skin surface is flattened, and scaling is reduced.[51] Patient, family, caregiver instruction on cleansing, environmental factors, and hydration is also important (see Patient Teaching tips).

Practice Point

Skin can become dry when TEWL drops below 10%.

Patient Teaching

To avoid xerosis, patients should be given clear instructions regarding cleansing, their environment, and remaining hydrated.

Cleansing

Instruct the patient to:

- avoid long baths (<15 minutes) or consider showering instead
- bathe every other day rather than daily
- use tepid water rather than hot water
- use pH-balanced soaps (4.0 to 6.5), avoid excessive use of deodorant soaps, and rinse well
- avoid vigorously using a washcloth to clean the skin
- pat or blot the skin, rather than rubbing with a towel, so some water is left on the skin
- apply moisturizers *immediately* after bathing or showering.

Environmental

Instruct the patient to:

- use a humidifier during the winter months when central heating is being used
- drink plenty of water
- wear a sunscreen with a sun protection factor (SPF) of 15 or higher that contains a moisturizer
- use nonfragrant laundry detergents, fabric softeners, and products.

Hydration

Instruct the patient to:

- use moisturizers, applying frequently, using correct gentle application technique for specific product (check product directions on how to use), and implementing fall safety

precautions because bathing surfaces may be slippery if using bath oils.

Practice Point

Stop the xerosis cycle!

- Dry skin
- Pruritus
- Scratching

Pruritus

Pruritus is the medical term for itchy skin and is a common symptom for several diseases. Therefore, taking a detailed history aids in determining if the cause is from such an underlying disease or if it's just untreated xerosis. For example, pruritus may be a symptom of renal or liver disease, scabies, or dry skin from aging (Table 4-6). Helping patients understand the itch–scratch–itch cycle and their own behavioral pattern is important to successful management. Because many moisturizers dissipate after 3 to 4 hours, these authors recommend using long-lasting moisturizers to cool, soothe, and restore barrier function. The goal is to break the itch–scratch–itch cycle, which happens because dry skin is often itchy, causing patients to scratch their skin (Table 4-7). In turn, excessive scratching can ultimately lead to a break in the skin. Once the skin barrier is broken, it becomes a portal of entry for bacteria, which can lead to infection. This repetitive scratching causes chronic thickening of the dermis known as lichenification. Therefore, prompt identification of the itch–scratch–itch cycle as well as teaching the patient about skin damage (from scratching) is extremely important in helping the skin to heal and reducing the occurrence of lichenification (Fig. 4-7). Treating the person with pruritus incorporates many of the elements in Table 4-8.[35]

Table 4-6 Moisturizer Functions and Ingredients

Moisturizer Functions

- *Humectants* promote water retention within the stratum corneum.
- *Occlusives* minimize water loss to the external environment.
- *Emollients* contribute to stratum corneum hydration.

Moisturizer Ingredients (Main Types)

- Humectants
  - Glycerin
  - Urea
  - Hydroxy acids (lactic acid)
  - Propylene glycol
  - Emollients
  - Protein rejuvenators
- Occlusives and emollients
  - Petrolatum
  - Mineral oil
  - Lanolin

Table 4-7 Pruritus

Pruritus is frequently seen in the following conditions and diseases:

- Brain tumor
- Biliary cirrhosis
- Diabetes mellitus
- Drugs
- Idiopathic (has no diagnostic cause)
- Liver disease
- Malignancies (especially in prevalent Hodgkin's where one-third of patients may have itching lasting up to 1 year)
- Multiple sclerosis
- Polycythemia (itch occurs after a hot bath)
- Psychological (anxiety disorders)
- Renal failure
- Senile pruritus (idiopathic pruritus in the elderly)
- Thyroid disorders (improves with treatment)
- Topical infections

Adapted with permission from Tomic-Canic, M. "Keratinocyte Cross-Talks in Wounds." *Wounds* 17:S3-6, 2005.

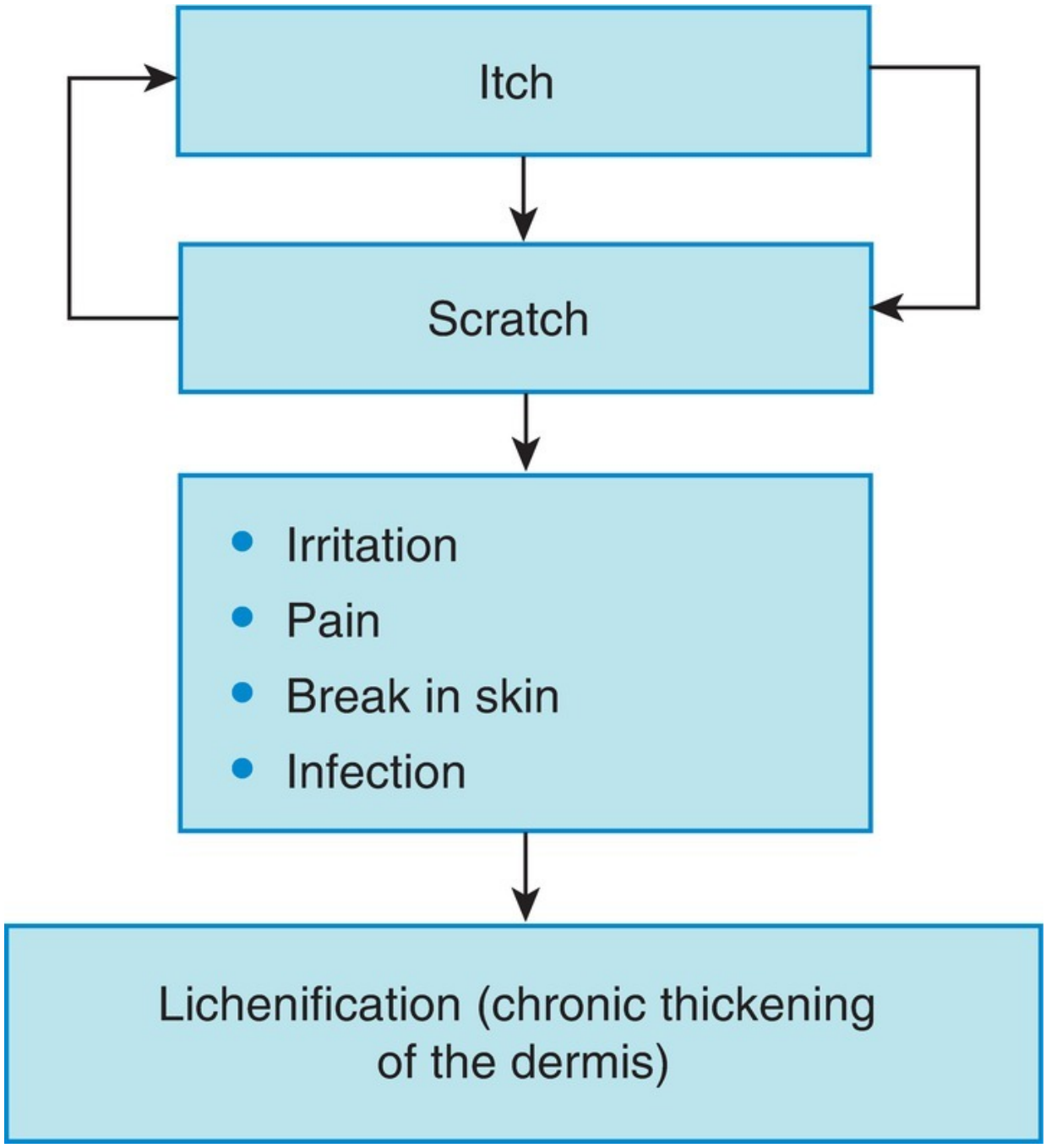


Figure 4-7. Itch–scratch–itch cycle.

Table 4-8 Treatment Plan for Pruritus

- Manage the underlying disease that causes the pruritus.
- Use topical emollients and bathing strategies as outlined in the xerosis care plan.
- Implement behavior modification (stop scratching and break the itch–scratch–itch cycle).
- Keep nails short.
- Wear gloves at night to decrease skin damage.
- Use cotton sheets, which may be more soothing to itchy skin.
- Help the patient avoid wearing clothing that can irritate the skin, such as wool or other "scratchy" fabrics.
- Limit the indiscriminate use of topical steroids and antihistamines as their effectiveness needs further investigation.

From Venna, S.S., Gilchrest, B.A. "Skin Aging and Photoaging." *Skin and Aging* 12:56-69, 2004; Gilhar, A., et al. "Ageing of Human Epidermis: The Role of Apoptosis, Fas and Telomerase." *The British Journal of Dermatology* 150(1):56-63, 2004.

Patient Teaching

Teach the patient about the itch–scratch–itch cycle.

Turgor

Turgor reflects the skin's resiliency and elasticity and hydration status. The assessment of skin turgor is used clinically to determine the extent of hydration, or fluid loss, in the body's tissue. The measurement is done by gently pinching up a skinfold (often on the back of the hand or the sternum region) between thumb and index finger so that it is raised for 3 seconds (Fig. 4-8). The skin is then released to observe how fast it returns to its normal position. If the skin remains elevated (tented) longer than 3 seconds, turgor is documented as decreased. With moderate to severe dehydration, decreased skin turgor causes the skinfold to remain elevated and only slowly return to its normal position. If no dehydration is present, the skin should return quickly to its normal position. Skin turgor also can be decreased in the elderly population as the skin loses its elasticity with aging. It is better to use the sternum region to assess skin turgor in the elderly.

A

Figure 4-8. Assessing skin turgor. **(A)**"Tent" the skin. **(B)** Normally, skin returns to its original state quickly, but (as shown) a slow return to its original shape may indicate dehydration or effect of aging. (From Ayello, E.A., Sibbald, R.G., Quiambao, P.C.H., et al. "Introducing a Moisture-Associated Skin Assessment Photo Guide For Brown Pigmented Skin." *World Council of Enterostomal Therapists Journal* 34(2):18-25, 2014. ©Ayello and Sibbald, used with permission.)

The deposit of fibrin into the skin especially on the lower legs will also decrease skin turgor. Advanced venous disease usually has lower leg varicosities, pitting edema of the ankles at the end of the day, and

exudation of fibrin on the lower leg starting in the gaiter area and spreading up on the calf towards the knee. This change is known as lipodermatosclerosis. Advanced disease will have a woody fibrosis that results in the skin losing its turgor. The clinician cannot tent the skin between the index finger and thumb. When the lipodermatosclerosis spreads to the dorsum of the foot, and you cannot pinch the skin over the dorsum of the second toe between the thumb and index finger (Stemmer's sign), it is an indication of lymphedema. If this is associated with venous disease and the lymphedema is secondary, always put the primary condition first in the description (venous lymphedema).[52]

Skin Integrity

Skin assessment requires the clinician to identity and document if the skin is intact, the presence of skin lesions, or a wound. Document any alteration that you may see such as skin lesions, scars, bruising, senile purpura, hemosiderin deposits, or edema noted (see Figs. 4-5 and 4-9).

Figure 4-9. Hemosiderin deposit.

Nutrition plays an important part in keeping the skin healthy and intact. Vitamin and/or mineral deficiencies can result in changes in the skin. Most vitamin deficiencies show up on the skin. “A lack of vitamin A can cause skin dryness and dry lips. B6 can leads to skin ulcers around the corners of the mouth, B7 can cause eczema, and B12 can cause vitiligo (hypopigmentation). Both Vitamins C and E are

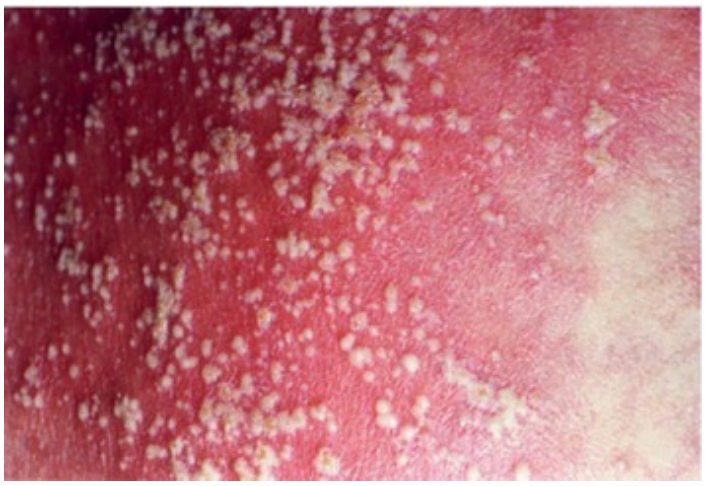
considered antioxidants, and deficiencies can cause premature skin aging, wrinkles, and dryness. Lack of vitamin D can contribute to psoriasis”[53,54] (see Chapter 10 Nutrition).

Primary and Secondary Skin Lesions

A skin lesion is a part of the skin that has an abnormal growth or appearance compared to the skin around it.

Two categories of skin lesions exist: primary and secondary. Primary skin lesions are abnormal skin conditions present at birth or acquired over a person's lifetime. Secondary skin lesions are the result of irritated or manipulated primary skin lesions. For example, if someone scratches a mole until it bleeds, the resulting lesion, a crust, is now a secondary skin lesion.

If a lesion is identified during initial examination, use the correct terminology (Fig. 4-10). When describing skin lesions, it is best to use consistent terms especially those used in dermatology. Lightly palpate any lesions to detect tenderness, firmness, and depth. Measure the length, width, and depth using a wound ruler or other wound dimension–measuring device.



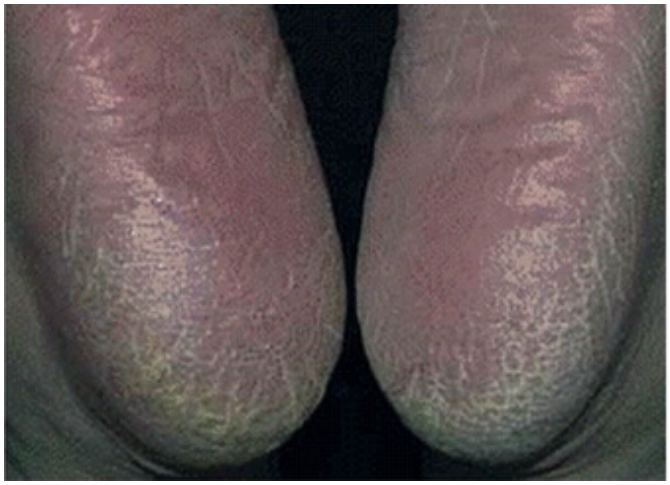


Figure 4-10. Primary and secondary skin lesions chart. (Used with permission from: (1) Ayala, C., Spellberg, B. *Boards and Wards for USMLE Step 2*, 2017. (2) Elder, D., Elenitsas, R., Rubin, A., et al. *Atlas and Synopsis of Lever's Histopathology of the Skin*, 2012. (3) Gru, A., Schaffer, A. *Hematopathology of the Skin*, 2016. (4) McConnell, T. *Nature of Disease*, 2013. (5) Neil Crowson, A., Magro, C. *Biopsy Interpretation of the Skin*, 2018. Wound, Ostomy and Continence Nurses Society, Wound, Ostomy and Continence Nurses Society® Core Curriculum: Wound Management, 2015. (6) Chung, E, Atkinson-McEvoy, L., Lai, N., et al. *Visual Diagnosis and Treatment in Pediatrics*, 2014. (7) The Podiatry Institute, Southerland, J., Alder, D., Boberg, J., et al. *McGlamry's Comprehensive Textbook of Foot and Ankle Surgery*, 2-Volume Set, 2012. (8) Requena, L., Requena, L., Kutzner, H. *Cutaneous Soft Tissue Tumors*, 2014.)

Scars

A scar is an area of fibrous tissue that replaces normal skin after an injury. Scars result from the biological process of wound repair in the skin as well as in other organs and tissues of the body. Thus, scarring is a natural part of the healing process. Assess for cause, location, appearance (color and size), and degree of tenderness. When examining scars on the leg area, look for a white porcelain satellite scar with peripheral telangiectasia and hyperpigmentation (see Fig. 4-11). Called atrophie blanche (AB), it is a common clinical sign in patients with healed leg ulcers, which may be a consequence of venous hypertension with local involvement or part of a livedoid vasculopathy (LV).[55]

Figure 4-11. Atrophie Blanche. (Courtesy of G. Sibbald. Used with permission.)

With the exception of very minor lesions, every wound (e.g., after accident, disease, or surgery) results in some degree of scarring. Figure 4-12 is an example of a healed surgical wound. Remember that scar tissue is vulnerable to injury.

Figure 4-12. (A) Surgical repair of tendon right foot. **(B)** Healed scar 4 months post op. (Courtesy of S. Baranoski. Used with permission.)

Edema

Edema is abnormal swelling caused by accumulation of fluid in the tissues. Swelling is a larger collection of edema that most commonly occurs in the hands, arms, ankles, legs, and feet (Fig. 4-13). It is often linked to the venous or lymphatic systems but must be distinguished from a deep hive or swelling under the skin called angioedema. Critical physiologic factors leading to edema include hypoalbuminemia, congestive heart failure, venous insufficiency, and lymphatic obstruction. Hypoalbuminemia can be caused by poor nutritional status, liver disease, kidney disease, and other chronic illnesses.[52]

Figure 4-13. Assessment of foot edema. (©Ayello and Sibbald, used with permission.)

Edema may be generalized or local. It can appear suddenly but usually develops subtly—the patient may first gain weight or wake up with puffy eyes. Generalized edema is known as “anasarca” and can be difficult to correct. Many patients wait until symptoms are well advanced before seeking medical help.

Assess area for edema by comparing one extremity to another. Press firmly with fingertip, usually index finger, on edematous site for 5 seconds and release (Fig. 4-14). Edema is “pitting” when it does not return to its normal contour rapidly.[48] Document the amount of pitting edema that you observe. There is no consensus in how edema is documented. Some use descriptive words such as mild, moderate, or severe, while others use numerical and plus signs (+1, +2, +3, +4); follow your facility policy. Elevate if condition warrants, use care in moving and repositioning edematous extremities, and use appropriate skin care products. Chapter 15 also includes more advanced information about edematous conditions.

A

B

Figure 4-14. Assessing pitting edema. **(A)** Examine lower leg. **(B)** Using your finger, depress the skin. **(C)** Remove your finger; note dent in the skin to access edema. (Ayello, E.A., Sibbald, R.G., Quiambao, P.C.H., et al. “Introducing a Moisture-Associated Skin Assessment Photo Guide for Brown Pigmented Skin.” *World Council of Enterostomal Therapists Journal* 34(2):18-25, 2014. ©Ayello and Sibbald, used with permission.)

Practice Point

Assessing for pitting edema rating scale

1+ equals 2 mm depression

2+ equals 4 mm depression

3+ equals 6 mm depression

4+ equals 8 mm depression

Reference: Edema rating scale: https://allnurses.com/edema-rating-scale-t59210

Practice Point

Factors affecting skin resilience[36]:

- Aging of skin
- Critical illness
- Malnutrition and dehydration
- Excess moisture
- Skin conditions that cause dryness

Nail Assessment for Onychomycosis

When doing a skin assessment, it is important to also look at the nails. Onychomycosis (tinea unguium) is a common fungal infection of the nail especially found in older persons.

It is typically caused by a dermatophyte type of fungus. Usually seen on the feet, it can also be on the nails on the hand. The nail becomes discolored (white, yellow, brown), thickens, and may crumble at the edge (see Fig. 4-15). Besides the esthetic cosmetic disfigurement of the nail, it can also be painful. In 2017, the International Diabetes Federation revised its recommendation to include the presence of onychomycosis in persons with diabetes and increases their risk of problems with their feet.[56] Doyle and colleagues' research supports that persons with diabetes who also have onychomycosis have a higher rate (as high as threefold) of foot ulceration and gangrene than those without onychomycosis.[57]

A

Figure 4-15. (A, B) Onychomycosis, nail fungus. (©Dr. R. Gary Sibbald, used with permission.)

Treatment for onychomycosis can consist of topical or oral antifungal medications.

Patient Teaching

Prevention of onychomycosis should include proper daily foot hygiene including washing the feet, wearing cotton socks and breathable shoes.

Skin Failure

We've discussed throughout this chapter that skin is the largest organ of the body. Can the skin as an organ fail? The literature supporting skin failure is very limited.[58–60] It is covered in more detail in Chapter 13. As an organ that requires oxygen and nutrients to maintain function, skin is subject to failure similar to any other organ. Skin failure can occur concomitantly with multiorgan system failure in critical care situations or at the end of life as a component of the dying process. The topic of skin failure is controversial and is closely related to topic of pressure injury avoidability.[58]

Summary

The skin is the largest organ of the body and commonly the most forgotten. Skin is exposed daily to environmental irritants and chemicals as well as physical and mechanical injury, any of which may lead to impaired skin integrity. This chapter provided an overview of skin structure and criteria for basic skin assessment including nails. The importance of identifying common skin conditions, specifically xerosis and pruritus, including the role of breaking the itch–scratch–itch cycle, is described. The use of moisturizers in their treatment is also highlighted. Skin assessment needs to include looking at the nails for fungal infection as well as the presence of any scars. Other skin conditions can be found in Chapter 13.

PATIENT SCENARIO

Clinical Data

A 20-year-old female patient presented to the office with thickened nails along with painful, cracked, discolored and scaly skin. She admits to going for frequent pedicures at a local salon. She feels that may be the source of her fungal condition. The dermatologist has treated her skin and she had been self-treating with topical antifungal medications with no success. Her diagnosis was confirmed as Onychomycosis.

Her physical exam was significant for excessive scaly skin, brittle and thickened nails. (See Figure 4-15). During her assessment she admitted that she is not consistent in applying the topical medication.

Case DiscussionThe nurse educated her on the correct application of the antifungal solution and the importance of continued application if her fungal infection was to be alleviated. She was started on our typical treatment protocol of topical medication. She returned demonstrated the application of the topical antifungal polish to her nails. She was also instructed on applying topical antifungal cream twice daily, and used antifungal shoe spray in her shoes twice

weekly. After six months of continued therapy she returned to the office. Her skin was no longer scaly or discolored and her fungal infection was gone.

Show What You Know

1. **While bathing a patient, you notice some flakes of skin on the washcloth. Which layer of the skin is this?**
   A. Stratum granulosum
   B. Stratum spinosum
   C. Stratum lucidum
   D. Stratum corneum

2. **Which one of the following is a normal function of the skin?**
   A. Synthesis of vitamin K
   B. Elimination of carbon dioxide
   C. Regulation of glucose levels by the Langerhans cells
   D. Thermal regulation by skin blood flow dilation or constriction

3. **What is the role of keratinocytes in skin?**
   A. Differentiation
   B. Cross-talk to fibroblasts
   C. Participating in BMZ
   D. Maintenance and repair of the barrier

4. **Which one of the following is NOT considered part of a routine skin assessment?**
   A. Color
   B. Turgor
   C. Temperature
   D. Ankle–brachial index (ABI)

5. **Which one of the following should be included in the care plan of a person with xerosis?**
   A. Have the patient shower daily.
   B. Use a deodorant soap.
   C. Dry the skin completely with vigorous rubbing.
   D. Apply an emollient immediately after bathing.

6. **Which of the following best defines pruritus?**
    A. Multiple blisters on the skin
    B. Traumatic open area on the skin
    C. Itchy skin
    D. Weepy skin

7. **In assessing a patient's skin, you find a raised area larger than 1 cm that is filled with serous fluid. This should be correctly documented as a:**
    A. papule.
    B. vesicle.
    C. pustule.
    D. bulla.

Answers to these questions are found on pages 681 and 682.

References

1. Orsted, H.L., Keast, D.H., Forest-Lande, L., et al. Foundations of Best Practice for Skin and Wound Management. Skin: Anatomy, Physiology and Wound Healing. Wounds Canada. Canadian Association of Wound Care. 2017. Available at: https://www.woundscanada.ca/docman/public/health-care-professional/bpr-workshop/166-wc-bpr-skin-physiology/file
2. Hill, M.A. Embryology. Main Page. January 27, 2019. Retrieved from https://embryology.med.unsw.edu.au/embryology/index.php/Main_Page
3. Koch, S., et al. “Skin Homing of Langerhans Cell Precursors: Adhesion, Chemotaxis, and Migration.” The Journal of Allergy and Clinical Immunology 117:163-8, 2006.
4. Blumenberg, M., Tomic-Canic, M. “Human Epidermal Keratinocyte: Keratinization Processes.” EXS 78:1-29, 1997.
5. Freedberg, I.M., et al. “Keratins and the Keratinocyte Activation Cycle.” The Journal of Investigative Dermatology 116:633-40, 2001.
6. Tomic-Canic, M., et al. “Epidermal Repair and the Chronic Wound.” In D.T. Rovee, H.I. Maibach, eds. The Epidermis in Wound Healing. Boca Raton, FL: CRC Press LLC, 2004:25-57.
7. Tomic-Canic, M. “Keratinocyte Cross-Talks in Wounds.” Wounds 17:S3-6, 2005.
8. Morasso, M.I., Tomic-Canic, M. “Epidermal Stem Cells: The Cradle of Epidermal Determination, Differentiation and Wound Healing.” Biology of the Cell 97:173-83, 2005.
9. Jensen, J.M., Proksch, E. The skin’s barrier. Giornale Italiano di Dermatologia e Venereologia 144(6):689-700, December 2009.
10. Habif, T.P. Clinical Dermatology: A Color Guide to Diagnosis and Therapy. Philadelphia, PA: Mosby, 2004.
11. Kanitakis, J. “Anatomy, Histology and Immunohistochemistry of Normal Human Skin.” European Journal of Dermatology 12:390-9; quiz 400, 2002.
12. Eckes, B., Krieg, T. “Regulation of Connective Tissue Homeostasis in the Skin by Mechanical Forces.” Clinical and Experimental Rheumatology 22:S73-6, 2004.
13. Yosipovitch, G., Hu, J. “The Importance of Skin pH.” Skin and Aging 11:88-93, 2003.
14. Waller, J.M., Maibach, H.I. “Age and Skin Structure and Function, A Quantitative Approach (I): Blood Flow, pH, Thickness, and Ultrasound Echogenicity.” Skin Research and Technology 11:221-35, 2005.
15. Rippke, F., et al. “Stratum Corneum pH in Atopic Dermatitis: Impact on Skin Barrier Function and Colonization with *Staphylococcus aureus*.” American Journal of Clinical Dermatology 5:217-23, 2004.

16. Yilmaz, E., Borchert, H.H. “Effect of Lipid-Containing, Positively Charged Nanoemulsions on Skin Hydration, Elasticity and Erythema—An In Vivo Study.” International Journal of Pharmaceutics 307:232-8, 2006.
17. Kurabayashi, H., et al. “Inhibiting Bacteria and Skin pH in Hemiplegia: Effects of Washing Hands with Acidic Mineral Water.” American Journal of Physical Medicine & Rehabilitation 81:40-6, 2002.
18. Damjanov, I. Pathology for the Health-Related Professions. Philadelphia, PA: W.B. Saunders, 2000.
19. Ayello, E.A., Sibbald, R.G., Quiambao P.C.H., et al. “Introducing a Moisture-Associated Skin Assessment Photo Guide for Brown Pigmented Skin.” World Council of Enterostomal Therapists Journal 34(2):18-25, 2014.
20. Yamasaki, K., Gallo, R.L. “Antimicrobial Peptides in Human Skin Disease.” European Journal of Dermatology 18(1):11-21, 2008.
21. Hughes, E., Van Onselen, J. Dermatology Nursing: A Practical Guide. London, UK: Churchill Livingstone Inc., 2001.
22. Bardan, A., et al. “Antimicrobial Peptides and the Skin.” Expert Opinion on Biological Therapy 4:543-9, 2004.
23. Niyonsaba, F., Ogawa, H. “Protective Roles of the Skin Against Infection: Implication of Naturally Occurring Human Antimicrobial Agents Beta-Defensins-Cathelicidin LL-37 and Lysozyme.” Journal of Dermatological Science 40:157-68, 2005.
24. Barakat, R., Oakley, O., Him, H., et al. Extra-gonadal Sites of Estrogen Biosynthesis and Function. BMB Reports 49(9):488-96, 2016.
25. Stante, M., et al. “Itch, Pain, and Metaesthetic Sensation.” Dermatology and Therapy 18:308-13, 2005.
26. Charkoudian, N. “Skin Blood Flow in Adult Human Thermoregulation: How It Works, When It Does Not, and Why.” Mayo Clinic Proceedings 78:603-12, 2003.
27. Minson, C.T. “Hypoxic Regulation of Blood Flow in Humans. Skin Blood Flow and Temperature Regulation.” Advances in Experimental Medicine & Biology 543:249-62, 2003.
28. Kupper, T.S., Fuhlbrigge, R.C. “Immune Surveillance in the Skin: Mechanisms and Clinical Consequences.” Nature Reviews Immunology 4:211-22, 2004.
29. Wolpowitz, D., Gilchrest, B.A. “The Vitamin D Questions: How Much Do You Need and How Should You Get It?” Journal of the American Academy of Dermatology 54:301-17, 2006.
30. Barrientos, S., Stojadinovic, O., Golinko, M.S., et al. “Growth Factors and Cytokines in Wound Healing.” Wound Repair and Regeneration 16(5):585-601, 2008.
31. Poljšak, B., Dahmane, R.G., Godić, A. “Intrinsic Skin Aging: The Role of Oxidative Stress.” Acta Dermatovenerol Alp Pannonica Adriat 21:33-6, 2012.

32. Vierkötter, A., Krutmann, J. “Environmental Influences on Skin Aging and Ethnic-Specific Manifestations.” Dermato-endocrinology 4(3):227-31, 2012.
33. Epel, E.S., Blackburn, E.H., Lin, J., et al. “Accelerated Telomere Shortening in Response to Life Stress.” Proceedings of the National Academy of Sciences 101(49):17312-5, 2004.
34. Venna, S.S., Gilchrest, B.A. “Skin Aging and Photoaging.” Skin and Aging 12:56-69, 2004.
35. Gilhar, A., et al. “Ageing of Human Epidermis: The Role of Apoptosis, Fas and Telomerase.” The British Journal of Dermatology 150(1):56-63, 2004.
36. Dealy, C. “Skin Care and Pressure Ulcers.” Advances in Skin & Wound Care 22(9):421-8, quiz 429-30, 2009.
37. Waller, J.M., Maibach, H.I. “Age and Skin Structure and Function, A Quantitative Approach (II): Protein, Glycoaminoglycan, Water, and Lipid Content and Structure.” Skin Research and Technology 12:145-54, 2006.
38. Zouboulis, C.C., Makrantonaki, E. “Hormonal Therapy of Intrinsic Aging.” Rejuvenation Research 15(3):302-12, 2012.
39. Ashworth, J.J., Smyth, J.V., Pendleton, N., et al. “Polymorphisms Spanning the *0N* Exon and Promoter of the Estrogen Receptor-Beta (ERB) Gene *ESR2* Are Associated with Venous Ulceration.” Clinical Genetics 73(1):55-61, 2008.
40. Gilliver, S.C., Ashcroft, G.S. “Sex Steroids and Cutaneous Wound Healing: The Contrasting Influences of Estrogens and Androgens.” Climacteric 10(4):276-88, 2007.
41. Fletcher, K. “Skin: Geriatric Self-learning Module.” MEDSURG Nursing 14(2):138-42, 2005.
42. Lee, B., Tomic-Canic, M. “Tissue Specificity of Steroid Action: Glucocorticoids in Epidermis.” In M. Krstic-Demonacos, C. Demonacos, eds. Molecular Mechanisms of Action of Steroid Hormone Receptors. Kerala, India: Research Signpost, 2002.
43. Matsumura, Y., Anathaswamy, H.N. “Toxic Effects of Ultraviolet Radiation on the Skin.” Toxicology and Applied Pharmacology 195(3):298-308, 2004.
44. Fisher, G.J. “The Pathophysiology of Photoaging of the Skin.” Cutis 75:5-8; discussion 8-9, 2005.
45. Geriatric Nursing Resources for Care of Older Adults. Normal Aging Changes. Available at: http://GeronurseOnline.org. Accessed June 6, 2006.
46. What Are the ABCD Test of Moles for Melanoma. Available at: https://www.Reference.com/health/abcd-test-moles-melanoma-f323b1637916c640
47. Baranoski, S. “Integumentary Anatomy: Skin, the Largest Organ.” In J. McCulloch, L. Kloth, eds. Wound Healing Evidence Based Management, 4th ed. Philadelphia, PA: EA Davis Co., 2010:1-8. Chap. 1.
48. Thomas-Hess, C. “Performing a Skin Assessment.” Nursing 40(7):66, 2010.
49. Sommers, M.S. “Making Sense of Skin Color in Clinical Care.” Clinical Nursing Research 12(4):495-516, 2012.

50. Skin Inc. The Fitzpatrick Skin Type Classification Scale. November 2007. Available at: http://www.skininc.com/skinscience/physiology/10764816.html. Last accessed June 2, 2014.
51. Lebwohl, M., et al. Treatment of Skin Disease: Comprehensive Therapeutic Strategies. London, UK: Harcourt Publishers Limited (Mosby), 2002.
52. Raju, S., Furrh, J.B., Neglen, P. Diagnosis and Treatment of Venous Lymphedema. Journal of Vascular Surgery 55(1):141-9, 2012.
53. Kaminski, M.V., Drinane, J.J. Learning the Oral and Cutaneous Signs of Micronutrient Deficiencies. Journal of the Wound Ostomy and Continence Nursing 4(12):127-35, 2014.
54. Curejoy Editorial. 9 Vitamin Deficiencies That Show Up On Your Skin. Available at: http://www.curejoy.com/content/vitamin-deficiencies-that-show-up-on-your-skin/. Accessed May 2, 2019.
55. Alavi, A., Hafner, J., Dutz, J.P., et al. Atrophie Blanche: Is It Associated with Venous Disease or Livedoid Vasculopathy? Advances in Skin & Wound Care 27:518-24; quiz 525-6, 2014.
56. International Diabetes Federation. Clinical Practice Recommendation on the Diabetic Foot: A Guide for Health Care Professionals: International Diabetes Federation, 2017. Available at: file:///C:/Users/eliza/Downloads/IDF_DF_Foot_CPR_2017_Final%20(1).pdf
57. Doyle, J., Boyko, W., Ryu, S., et al. Onychomycosis Among Diabetic Patients: Prevalence and Impact of Nonfungal Foot Infections. Diabetes 49(Suppl 1): A195-6, 2000.
58. Levine, J.M. Skin Failure: An Emerging Concept. Journal of the American Medical Directors Association 17:666-9, 2016.
59. Levine, J.M. Unavoidable Pressure Injuries, Terminal Ulceration, and Skin Failure: In Search of a Unifying Classification System. Advances in Skin & Wound Care 30(5):200-2, 2017.
60. Ayello, E.A., Levine, J.M., Langemo, D., et al. Reexamining the Literature on Terminal Ulcers, SCALE, Skin Failure, and Unavoidable Pressure Injuries. Advances in Skin & Wound Care 32(3):109-21, 2019.

5

Acute and Chronic Wound Healing

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We gratefully acknowledge the contributions of Keith Harding MB, ChB, FRCGP, FRCP, FRCS for his work on previous editions of this chapter.

Objectives

After completing this chapter, you will be able to:

- describe the physiology of normal and pathophysiology of abnormal wound healing
- discuss the cascade of wound healing events
- compare and contrast acute and chronic wound healing
- appraise terminology used in clinical practice to refer to a chronic wound
- identify host factors that affect wound healing
- discuss the effects of biofilms on wound healing
- evaluate the key consensus statements for the assessment and treatment of biofilms
- explain the principles of wound bed preparation and TIME in the management of patients with wounds
- describe symptoms and symptom clusters associated with chronic wounds.

Acute Wound Healing Events

When a patient experiences tissue injury, it's essential that hemostasis is rapidly achieved and tissue is repaired to prevent invasion by pathogens and restore tissue function. The process of wound healing is a complex sequence of events that starts when the injury occurs and ends with complete wound closure and successful, functional scar tissue organization. Although tissue repair is commonly described as a series of stages, in reality it's a continuous process during which cells undergo a number of complicated biological changes to facilitate hemostasis, combat infection, migrate into the wound space, deposit a matrix, form new blood vessels, and contract to close the defect.

However, wound closure isn't a marker of healing completion; the wound continues to change, in a process called remodeling, for up to 18 months post closure. During this prolonged phase of remodeling and maturation, the closed wound is still quite vulnerable.

Patient Teaching

Remind your patient that the process of wound healing can take up to 18 months. Although the wound may appear to be closed, changes are occurring in the underlying tissue. This means that the wound is still vulnerable to damage. Tell the patient to seek professional advice if he or she has any concerns about the wound.

Acute Wound Healing Cascade

The process of healing is usually divided into four phases—hemostasis, inflammation, proliferation/repair, and maturation/remodeling—each of which overlaps the others while remaining distinct in terms of time after injury (Fig. 5-1).

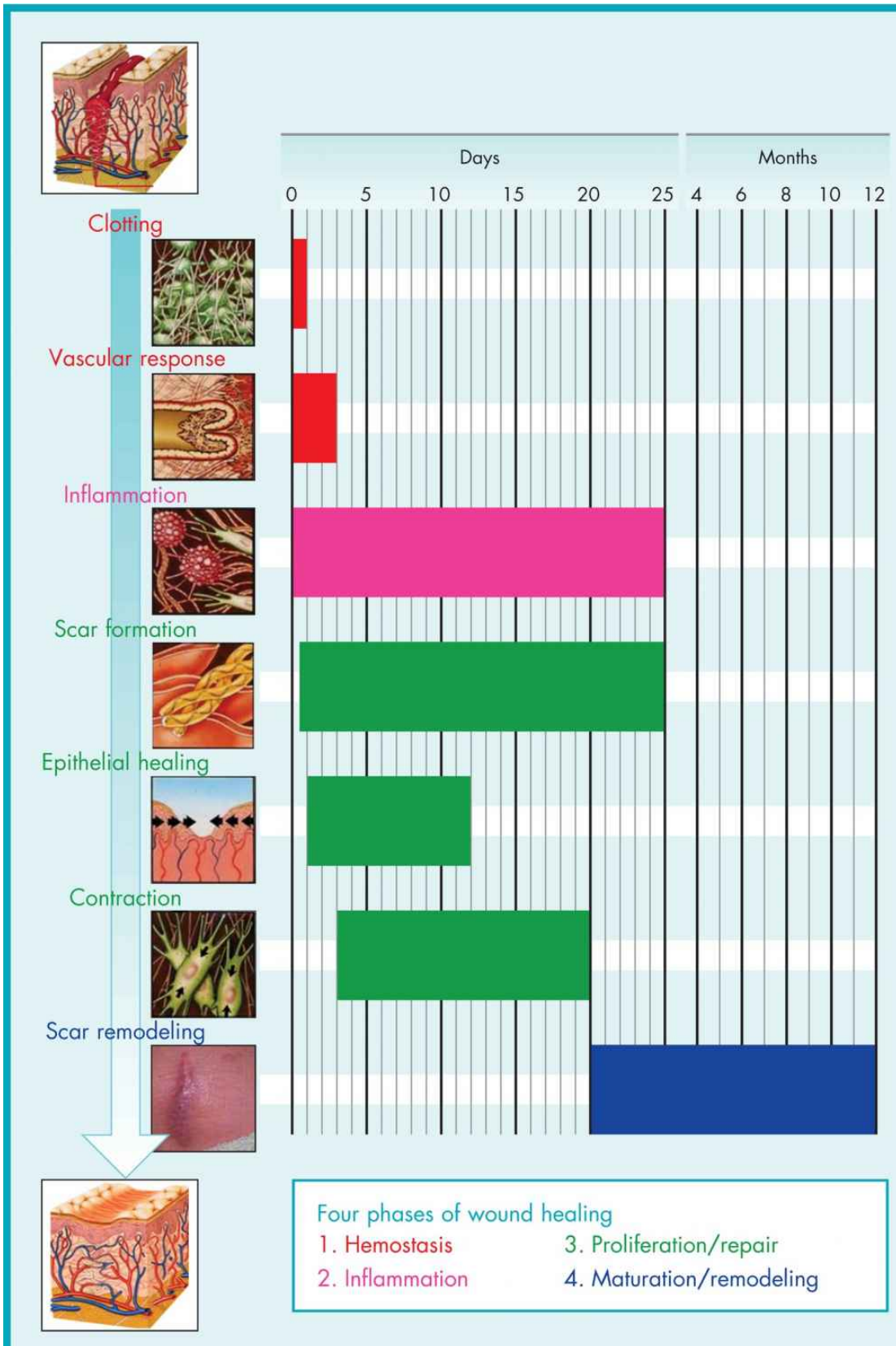

Days
Months
0
5
10
15
20
25
4
6
8
10
12
Clotting
Vascular response
Inflammation
Scar formation
Epithelial healing
Contraction
Scar remodeling
Four phases of wound healing
1. Hemostasis
2. Inflammation
3. Proliferation/repair
4. Maturation/remodeling

Figure 5-1. Sequence of molecular and cellular events in skin wound healing.

Patient Teaching

Inform your patient that the process of healing is often separated into four different phases. When a wound is being assessed, the clinician is looking for signs of progress as well as deterioration.

Hemostasis

The disruption of tissue following injury causes hemorrhage, which initially fills the wound and exposes the blood to various components of the extracellular matrix (ECM).[1] Platelets aggregate and degranulate, which activates factor XII (Hageman factor), resulting in clot formation and hemostasis. Hemostasis stops hemorrhage at the site of blood vessel damage. This is essential as it preserves the integrity of the closed and high-pressure circulatory system to limit blood loss. A fibrinous clot forms during coagulation, acting as a preliminary matrix within the wound space into which cells can migrate.

After the fibrin clot forms, another mechanism is activated as part of the body's defense system—fibrinolysis—in which the fibrin clot starts to break down. This process prevents clot extension and dissolves the fibrin clot to allow ease of further cell migration into the wound space,[2] allowing the next stage of healing to proceed.

Inflammatory Phase

As the fibrin clot is degraded, the capillaries dilate and become permeable, allowing fluid into the injury site and activating the complement system. The complement system is composed of a series of interacting, soluble proteins found in serum and extracellular fluid that induce lysis and the destruction of target cells. C3b, a complement molecule, helps bind (opsonize) neutrophils to bacteria, facilitating phagocytosis and subsequent bacterial destruction.

Cytokines and some proteolytic fragments that are hemoattractive are also found in the wound space.[2] Their abundance and accumulation at the site of injury initiate a massive influx of other cells. The two main inflammatory cells—neutrophils and macrophages—are attracted to the wound space to mount an acute inflammatory response.[3]

Neutrophils appear in a wound shortly after injury and reach their peak number within 24 to 48 hours; their main function is to destroy bacteria by the process of phagocytosis. Neutrophils have a very short life span: after 3 days without infection, their numbers reduce rapidly.

Tissue macrophages are derived from blood monocytes and arrive approximately 2 to 3 days after injury, followed by lymphocytes. Like neutrophils, macrophages also destroy bacteria and debris through phagocytosis; however, macrophages are also a rich source of biological regulators, including cytokines and growth factors, bioactive lipid products, and proteolytic enzymes, which are also essential for the normal healing process.[2,4]

Cytokines, Growth Factors, and Chemotaxis

Cytokine is a broad term that includes such molecules as growth factors, interleukins, tumor necrosis factors, and interferons. These molecules act on a variety of cells by exerting a wide range of biological functions by means of their specific receptors on target cells or proteins. Pathogens, endotoxins, tissue degradation products, and hypoxia are all factors that stimulate cells to produce cytokines following injury. The main cellular sources for these cytokines are

platelets, fibroblasts, monocytes and macrophages, and endothelial cells. These cells are involved in physiological as well as pathological conditions (e.g., tumors), although in wound healing, they play an important role as mediators. Cytokines regulate cell proliferation, migration, matrix synthesis, deposition and degradation, and inflammatory responses in the repair process (Table 5-1).

Table 5-1 Major Cytokines Involved in Wound Healing

Cytokine	Cell Source	Biological Activity
Proinflammatory Cytokines		
Tumor necrosis factor-alpha (TNF-α)	Macrophages	↑ PMN margination and cytotoxicity ↑ MMP synthesis
Interleukin-1 (IL-1)	Macrophages, keratinocytes	↑ Fibroblast and keratinocyte chemotaxis ↑ MMP synthesis
Interleukin-6 (IL-6)	Macrophages, keratinocytes, PMNs	↑ Fibroblast proliferation
Interleukin-8 (IL-8)	Macrophages, fibroblasts	↑ Macrophage and PMN chemotaxis ↑ Collagen synthesis
Interleukin-γ	Macrophages, T lymphocytes	↑ Macrophage and PMN activation ↓ Collagen synthesis ↑ MMP synthesis
Anti-inflammatory Cytokines		
Interleukin-4 (IL-4)	T lymphocytes, basophils, mast cells	↓ TNF-α, IL-1, IL-6 synthesis ↑ Fibroblast proliferation, collagen synthesis
Interleukin-10 (IL-10)	T lymphocytes, macrophages, keratinocytes	↓ TNF-α, IL-1, IL-6 synthesis ↓ Macrophage and PMN activation

Immediately after injury, platelet degranulation releases numerous cytokines, including platelet-derived growth factor (PDGF), transforming growth factor (TGF), and epidermal growth factor (EGF). These cytokines, together with other chemotactic agents, such as tissue debris and pathogenic materials, attract neutrophils and, later, macrophages. In time, these cells contribute to a larger number and variety of cytokines, which participate in the healing process.[3] (See *Patient Teaching: Normal and abnormal signs of the inflammatory process*.)

Cytokines have diverse effects on the healing process, interacting in additive, synergistic, or inhibitory ways (Table 5-2). For example, keratinocyte growth factor enhances the stimulation of collagenase

synthesis exerted by insulin-like growth factor. TGF is inhibitory to fibroblast growth in the presence of EGF but stimulates cell division when PDGF is present.

Table 5-2 Major Growth Factor Families

Growth Factor Family	Cell Source	Actions
Transforming growth factor (TGF)-β TGF-β1 TGF-β2 TGF-β3	Platelets Fibroblasts Macrophages	Chemotactic for fibroblasts Promotes extracellular matrix formation ↑ Collagen and tissue inhibitors of metalloproteinase (TIMP) synthesis ↓ Matrix metalloproteinase (MMP) synthesis Reduces scarring ↓ Collagen ↓ fibronectin
Platelet-derived growth factor (PDGF) PDGF-AA; PDGF-BB; VEGF	Platelets Macrophages Keratinocytes Fibroblasts	Activates immune cells and fibroblasts Promotes extracellular matrix (ECM) formation ↑ Angiogenesis ↑ Angiogenesis
Fibroblast growth factor (FGF) Acidic FGF, basic FGF, KGF	Macrophages Endothelial cells Fibroblasts	↑ Angiogenesis ↑ Keratinocyte proliferation and migration ↑ ECM deposition
Insulin-like growth factor (IGF) IGF-I, IGF-II, insulin	Liver Skeletal muscle Fibroblasts Macrophages Neutrophils	↑ Keratinocyte and fibroblast proliferation ↑ Angiogenesis ↑ Collagen synthesis ↑ ECM formation ↑ Cell metabolism
Epidermal growth factor (EFG) EGF, HB (heparin binding), TGF-α, amphiregulin, betacellulin	Keratinocytes Macrophages	↑ Keratinocyte proliferation and migration ↑ ECM formation
Connective tissue growth factor (CTGF)	Fibroblasts Endothelial cells Epithelial cells	↑ Collagen synthesis Mediates action of TGF-βs on collagen synthesis

Proliferation Phase

The proliferation phase usually begins 3 days after an injury and lasts for a few weeks. This phase is characterized by the formation of granulation tissue in the wound space. The new tissue consists of a matrix of fibrin, fibronectin, collagens, proteoglycans, glycosaminoglycans (GAGs), and other glycoproteins.[5] Fibroblasts move into the wound space and proliferate. Because the type III collagen in the wound has decreased tensile strength, the patient is at risk for such abnormalities as wound dehiscence or opening of wound edges in a previously closed wound that healed by primary intention. If organs are protruding from the now opened wound, it's called *evisceration*, which is a medical emergency that requires immediate surgery.

Patient Teaching Normal Signs of the Inflammatory Process

Discuss the *normal signs* of the inflammatory process with your patient:

- Redness
- Swelling
- Heat
- Pain

Clarify that in the early stages of the healing process, you would expect the wound to exhibit these signs. However, advise the patient to *seek urgent medical attention* if any of the signs listed below are present, as these may be *signs of infection:*

- Wound breakdown
- Bleeding
- Increased pain
- Pus or unusual drainage
- Spreading redness around the wound
- Flu-like symptoms

Practice Point

Keep in mind that the induration, heat, discomfort, redness, and swelling experienced during the inflammatory phase are part of the normal wound healing processes and aren't, at this stage, likely to be

due to wound infection. Remember to share this information with your patients.

Practice Point

During the first 3 weeks after surgery, the patient is at high risk for wound dehiscence and evisceration. Advise the patient that he or she should follow any postsurgical advice very carefully as the repaired tissue will not regain its full strength. The wound is at risk of breakdown if undue pressure is exerted on the area. For instance, patients who have undergone an abdominal procedure should support their abdomen with a soft pillow if they need to cough. They should also avoid any heavy lifting or exertion as designated by their physician.

Role of Fibroblasts

Fibroblasts play a key role during the proliferation phase, appearing in large numbers within 3 days of injury and reaching peak levels on the 7th day. During this period, they undergo intense proliferative and synthetic activity. Fibroblasts synthesize and deposit extracellular proteins during wound healing, producing growth factors and angiogenic factors that regulate cell proliferation and angiogenesis.[6]

Granulation tissue is composed of many mesenchymal and nonmesenchymal cells with distinct phenotypes, inflammatory cells, and new capillaries embedded in a loose ECM composed of collagens, fibronectin, and proteoglycans.

Role of ECM Proteins

ECM consists of proteins and polysaccharides and their complexes produced by cells in the wound space. The two main classes of matrix proteins are fibrous proteins (collagens and elastin) and adhesive

proteins (laminin and fibronectin). In addition, the ECM contains polysaccharides called proteoglycans and GAGs.

Collagen is the most abundant protein in animal tissue and accounts for 70% to 80% of the dry weight of the dermis.[7] The collagen molecule consists of three identical polypeptide chains bound together in a triple helix. Made mainly by fibroblasts, at least 19 genetically distinct collagens have been identified. Collagen synthesis and degradation are finely balanced.[2]

Elastin is a protein that provides elasticity and resilience.[8] It is composed of fibrous coils that stretch and return to their former shape, much like metallic coils. Because of these properties, elastin helps maintain tissue shape. Elastin represents only 2% to 4% of the human skin's dry weight; it's also in the lungs and blood vessels. It's secreted into the extracellular space as a soluble precursor, tropoelastin, which binds with a microfibrillar protein to form an elastic fiber network.

Laminin and fibronectin are two fiber-forming molecules. Their function is to provide structural and metabolic support to other cells. Fibronectin is found in plasma and contains specific binding sites on its molecular wall for cells, collagens, fibrinogens, and proteoglycans. It plays a central role in tissue remodeling, acting as a mediator for physical interactions between cells and collagens involved in ECM deposition, thereby providing a preliminary matrix.

Proteoglycans consist of a central core protein combined with a number of GAG chains that may be one or several types. GAGs consist of long, unbranched chains of disaccharide units that can range in number.[2] A highly complex group of molecules, proteoglycans, are characterized by their many diverse structural and organizational functions in tissue. Forming a highly hydrated gel-like "ground substance," they can contain up to 95% (w/w) carbohydrates. Originally, however, they were thought to contribute to tissue resilience due to their capacity to fill much of the extracellular space.

Angiogenesis

Angiogenesis is the formation of new vessels in the wound space and is an integral and essential part of wound healing.[9] The vascular endothelial cell (VEC) plays a key role in angiogenesis and arises from

the damaged end of vessels and capillaries. New vessels originate as capillaries, which sprout from existing small vessels at the wound edge. The endothelial cells from these vessels detach from the vascular wall, degrade and penetrate (invade) the provisional matrix in the wound, and form a knob-like or cone-shaped vascular bud or sprout. These sprouts extend in length until they encounter another capillary, to which they connect to form vascular loops and networks, allowing blood to circulate. This pattern of vascular growth is similar in skin, muscle, and intestinal wounds.

Epithelialization

Epithelial healing, or epithelialization, which begins a few hours after injury, is another important feature of healing. Marginal basal cells, which are normally firmly attached to the underlying dermis, change their cell adhesion property and start to lose their firm adhesion, migrating in a leapfrog or train fashion across the provisional matrix. Horizontal movement is stopped when cells meet. This is known as contact inhibition.

Wound Contraction

The final feature of the proliferation phase is wound contraction, which normally starts 5 days after injury. Wound contraction appears to be a dynamic process in which cells organize their surrounding connective tissue matrix, acting to reduce the healing time by reducing the amount of ECM that needs to be produced. The contractile activity of fibroblasts and myofibroblasts provides the force for this contraction. These cells may use integrins and other adhesion mechanisms to bind to the collagen network and alter its motility, bringing the fibrils and, subsequently, the wound edges closer.[2] Such contraction may not be important in a sharply incised, small, and noninfected wound; however, it's critical for wounds with large tissue loss.[10]

The myofibroblast theory suggests that the contraction force occurs when the movement of microfilament (actin) bundles (also termed *stress fibers*) contracts the myofibroblast in a muscle-like fashion. Because the myofibroblast displays many cell–cell and cell–matrix (fibronexus) contacts, the cellular contraction pulls collagen fibrils toward the body of the myofibroblast and holds them until they're

stabilized into position. This gathering of collagen fibers toward the myofibroblast cell “body” leads to the shrinkage of granulation tissue. The ECM of the wound is continuous with the undamaged wound margin, enabling the granulation tissue shrinkage to pull on the wound margin, leading to wound contraction. The myofibroblast theory further proposes that the coordinated contraction (cellular shortening) of many myofibroblasts, synchronized with the help of gap junctions, generates the force necessary for wound contraction.[6]

The traction theory proposes that fibroblasts bring about a closer approximation of matrix fibrils by exerting “traction forces” (analogous to the traction of wheels on tarmac) on ECM fibers to which they’re attached. This theory proposes that fibroblasts neither shorten in length nor act in a coordinated multicellular manner (as proposed by the myofibroblast theory); rather, a composite force, made up of traction forces of many individual fibroblasts, is responsible for matrix contraction. Such traction forces act as shearing forces tangential to the cell surface generated during cell elongation and spreading. According to the traction theory, the composite effect of many fibroblasts gathering collagen fibrils within the wound is thought to bring about wound contraction.[11]

Patient Teaching

Teach your patient who has a wound left open to heal (secondary intention) that clinicians will be looking for indications that wound healing is progressing normally:

- Healthy pink tissue in the wound bed
- Signs of new tissue growth at the wound edges
- Decreasing wound size over time

Maturation Phase

The maturation phase normally starts 7 days after injury and may last for 1 year or more. The initial component in the deposited ECM is fibronectin, which forms a provisional fiber network. Other components include hyaluronic acid and proteoglycans. The network has two main roles: as a substratum for the migration and growth of cells and as a template for subsequent collagen deposition. Collagen deposition becomes the predominant constituent of the matrix and soon forms fibrillar bundles and provides stiffness and tensile strength to the wound.

Collagen deposition and remodeling contribute to the increased tensile strength of skin wounds. Within 3 weeks of injury, the tensile strength is restored to approximately 20% of normal, uninjured skin. As healing continues, the skin gradually reaches a maximum of 70% to 80% tensile strength. Different organs regain tensile strengths to differing degrees. The remodeling process involves the balance between the synthesis and degradation of collagen. A range of collagenases regulates the latter. This process is also characterized by a gradual reduction in cellularity and vascularity.[3] Differentiation of fibroblasts into myofibroblasts with resultant apoptosis (programmed cell death) are also features of tissue remodeling.[12]

Practice Point

A patient history should always include information about prior wounds. Healed wounds never achieve the same tensile strength as uninjured skin, thereby increasing the potential for reinjury.

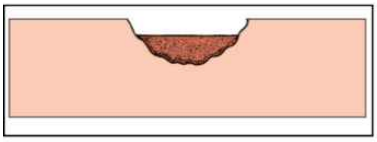
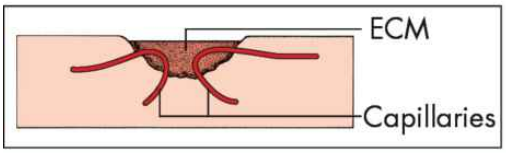
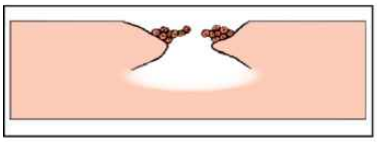
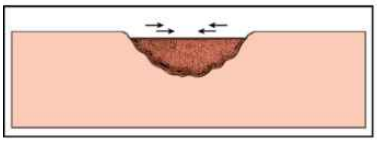
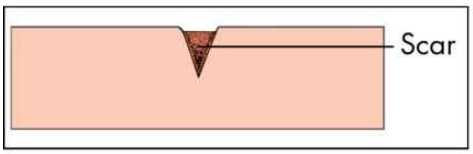
Patient Teaching

Remind the patient that the wounded area is never as strong as uninjured tissue so it is always vulnerable to damage. Simple measures such as keeping the scar tissue moisturized will help to optimize the condition of the tissue. Advise the patient to seek help if the scar begins to break down.

The scar is the final product of wound healing and is a relatively avascular and acellular mass of collagen that serves to restore tissue continuity and some degree of tensile strength and function. However, the strength of the scar remains less than that of normal tissue, even many years following injury, and it's never fully restored (Box 5-1).

Box 5-1 Summary of Wound Healing

The following is a summary of the events that occur during the phases of wound healing.

Hemostasis	
Platelets	Release cytokines (PDGF, TGF-β, EGF)
Inflammatory Phase	
Tissue debris and pathogens	Attract macrophages and neutrophils, which are responsible for: ● phagocytosis ● producing biological regulators, bioactive lipids, and proteolytic enzymes.
Proliferative Phase	
Fibroblasts	Responsible for: ● synthesizing and depositing extracellular proteins ● producing growth factors ● producing angiogenic factors.
ECM and granulation tissue 	ECM composed of: ● collagens and elastin ● adhesive proteins ● fibronectin and lamina ● polysaccharides ● proteoglycans ● glycosaminoglycans.
Angiogenesis 	Capillary growth into ECM
Reepithelialization 	Migration of marginal basal cells across the provisional matrix
Wound contraction 	Contraction of fibroblasts and myofibroblasts to bring wound edges closer
Maturation 	● Collagen deposition and remodeling ● Differentiation of fibroblasts into myofibroblasts with programmed cell death (apoptosis)
Resultant scar	Formation of a scar, an avascular, acellular mass of collagen

Practice Point

The width of the resultant scar of a wound healing by secondary intention is about 10% of the original defect, primarily due to the process of wound contraction, working in conjunction with proliferation.

Patient Teaching

Inform the patient of the changes that occur to scar tissue over time. Initially, the scar may be red and raised but over time will become paler and flatten out. This process may take up to 2 years. Occasionally, there may be signs of abnormal healing, such as a scar remaining raised or swollen. If this occurs, the patient should seek further advice from a healthcare practitioner as this may indicate hypertrophic or keloid scarring.

Role of Matrix Metalloproteinases in Wound Healing

Proteases, especially the matrix metalloproteinases (MMPs), play essential roles in all phases of normal wound healing (Box 5-2). For example, during the inflammatory phase, damaged ECM proteins (such as collagen) must be removed so that newly synthesized collagen molecules can correctly align with collagen molecules in the wound matrix, permitting migration of epidermal cells and fibroblasts into the wound bed (Box 5-3). To remove damaged collagen molecules, collagenases (Box 5-4), make a single cut in collagen molecules, which permits the gelatinases to further degrade collagen molecules into small fragments that are then removed from the injury area by neutrophils and macrophages. MMPs also play a key role in angiogenesis by first degrading the basement membrane that surrounds VECs. This causes new capillary buds to sprout and "channels" to erode the ECM, through which the VECs migrate, eventually creating new capillary arcs. Furthermore, MMPs are required for myofibroblasts to contract ECM during the maturation or remodeling phase. The actions of MMPs are controlled by their natural inhibitors, the tissue inhibitors of metalloproteinases (TIMPs).

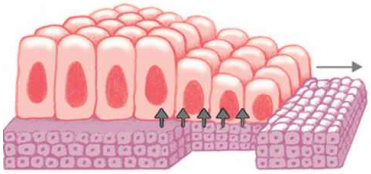
Box 5-2 Role of MMPs in Wound Healing

Proteases (especially MMPs) play important, beneficial roles in normal wound healing. They perform the following functions:

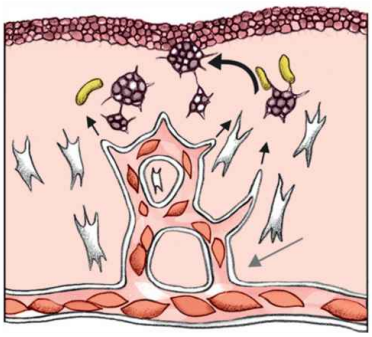
- Contract wound matrix through use of myofibroblasts
- Implement angiogenesis (breakdown of capillary basement membrane)
- Migrate cells (epidermal cells, fibroblasts, VECs)
- Remodel scar ECM
- Remove damaged ECM (especially during the inflammatory phase of healing)

Box 5-3 ECM Proteins and MMPs: Critical Factors for Epithelial Migration, Angiogenesis, and Contraction

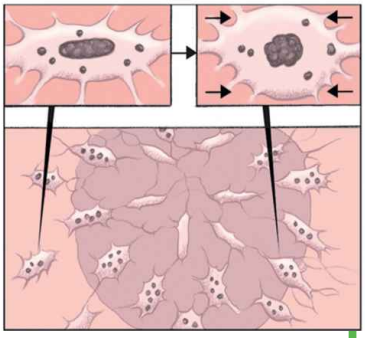
Epithelial migration. Epidermal cells at the leading edge of migrating sheets secrete several types of MMPs; fibroblasts migrating through provisional wound matrix also secrete MMPs.



Angiogenesis. Endothelial cells secrete MMPs that degrade the basement membrane surrounding capillaries, allowing endothelial cells to proliferate and migrate toward angiogenic factors produced by cells in ischemic areas.



Contraction. Fibroblasts transform into myofibroblasts, which express contractile fibers and MMPs, and as myofibroblasts contract, force is applied to collagen fibers that reduces the size of the wound.



Box 5-4 Families of MMPs, TIMPs, and ADAMs

Collagenases
- MMP-1, MMP-8, MMP-13, MMP-18
- Cut native type I collagen at one site

Gelatinases
- MMP-2, MMP-9
- Cut type collagen after collagenases make initial cut
- Cut native type IV collagen in basement membranes

Stromelysins
- MMP-3, MMP-10, MMP-11, MMP-19
- Cut core protein of proteoglycans

Metalloelastase/matrilysin
- MMP-7, MMP-12
- Cut multiple substrates, including type IV collagen

Membrane-type MMPs (MT-MMPs)
- MT-MMP-1 (MMP-14), MT-MMP-2 (MMP-15), MT-MMP-3 (MMP-16), MT-MMP-4 (MMP-17)
- Attached to plasma membrane, active pro-MMPs

Tissue inhibitors of metalloproteinases (TIMPs)
- TIMP-1, TIMP-2, TIMP-3, TIMP-4
- Specific inhibitors for MMPs

A disintegrin and metalloproteinase (ADAM)
- Aggrecanase-1 (ADAM-1)

Tumor necrosis factor-α (TNF-α)–converting enzyme (TACE)

Acute Versus Chronic Wound Healing

Molecular and Cellular Abnormalities in Chronic Wounds

There would appear to be little consensus regarding the definition of acute and chronic wound etiologies. Chronicity implies a prolonged or lengthy healing process, whereas acute implies uncomplicated, orderly or organized, or rapid healing. Bates-Jensen and Woolfolk[13] define an acute wound as “a disruption in the integrity of the skin and underlying tissues that progresses through the healing process in a timely and uncomplicated manner.” Typically, surgical and traumatic wounds, which heal by primary intention, are classified as acute.

On the other hand, Sussman[14] defines a chronic wound as “one that deviates from expected sequence of repair in terms of time, appearance, and response to aggressive and appropriate treatment.” The Wound Healing Society uses the definition of chronic wound as proposed in 1994 by Lazarus and colleagues: chronic wounds are wounds that “fail to progress through a normal, orderly, and timely sequence of repair or wounds that pass through the repair process without restoring anatomic and functional results”.[15] Chronicity is being said to occur when a wound does not heal within a 4-week period or in an orderly and timely manner with standard care.[16] Such wounds usually heal by secondary intention and are associated with pathology, for example, diabetes, vascular disease (venous leg ulcers) ischemic disease, pressure damage, and inflammatory diseases. However, with appropriate treatment, most venous leg ulcers should heal within 3 to 4 months,[17] so there is somewhat of a disconnect between the definition of a chronic wound in terms of duration versus how long it might take to heal. There are a number of different terms used to refer to a chronic wound (Box 5-5).

Box 5-5 Terminology Used to Describe a Chronic Wound

- Delayed healing[16]
- Nonhealing[16]

- Complex wound[17]
- Recalcitrant[18]
- Stalled[19]
- Hard-to-heal[20]

Teaching patients the words used to describe the types of wound healing (acute, chronic, primary, secondary) may help them appreciate the time it may take for their wound to heal.

Discuss the importance of moist wound healing to your patients, and inform them of what factors have been taken into consideration when deciding the most appropriate treatment for their wound.

Practice Point

Consider the terminology that is used in your own clinical practice, and reflect on how this might affect your assessment, documentation, and subsequent management of a patient with a wound.

The physiological differences between wounds that heal slowly and those that heal rapidly have been studied in a variety of ways (Box 5-6). One experiment explored the effect of chronic wound fluid on cell function.[21] Researchers cultured fibroblasts from human neonatal foreskin to use as a laboratory model of acute wounds. They then exposed the model to either chronic wound fluid or a control and found that chronic wound fluid dramatically inhibited the growth of the fibroblasts. According to Phillips et al.,[21] these results indicate that the microenvironment of chronic wounds impairs wound healing.

Box 5-6 Imbalanced Molecular Environments of Healing and Chronic Wounds

Healing Wounds	Chronic Wounds
• Functional extracellular matrix (ECM) • High mitogenic activity • Low inflammatory cytokines • Low protease, reactive oxygen species (ROS) • Mitotically competent cells • Low bioburden on the left hand side of the figure as another bullet point after "mitotically competent cells"	• Damaged ECM • High inflammatory cytokines • High protease, ROS • Low mitogenic activity • Senescent cells

Box 5-7 Factors Suggested to Affect Wound Healing

Local Factors[18]	• Oxygenation • Infection • Foreign body • Venous insufficiency
Systemic Factors[18]	• Age and gender • Sex hormones • Stress • Ischemia • Diseases: diabetes, keloids, fibrosis, hereditary healing disorders, jaundice, uremia • Obesity • Medications: glucocorticoid steroids, nonsteroidal anti-inflammatory drugs, chemotherapy • Alcoholism and smoking • Immunocompromised conditions: cancer, radiation therapy, AIDS • Nutrition
Biopsychosocial factors[51–57]	• Inflammation and sickness behaviors (pain, anxiety, depression, cognitive changes, sleep disturbances, anorexia)

Adapted from Guo, S., DiPietro, L.A. "Factors Affecting Wound Healing." *Journal Dental Research* 89(3):219-29, 2010.

Adapted from Guo, S., DiPietro, L.A. "Factors Affecting Wound Healing." *Journal Dental Research* 89(3):219-29, 2010.

Other researchers[22,23] theorize that prolonged inflammation is the most significant factor in delayed healing. Indeed, Hart[22] proposes that the prolonged inflammatory phase is due to the presence of

inflammatory leukocytes, typically neutrophils and their production of proinflammatory cytokines that perpetuate inflammation. He also argues that the release of tissue-damaging proteinases, which degrade newly formed tissue, delays or prevents normal wound healing processes. In addition to prolonged inflammation, Hart[22] suggests several other factors that may induce chronicity, including recurrent physical trauma, ischemic reperfusion injury, subclinical bacterial contamination, and foreign bodies.

Because chronic wounds are typically characterized by full-thickness tissue loss, reepithelialization is prolonged due to the loss of appendages.[14] Normally, epithelial cells require the smooth, moist surface of the basement membrane to move across the wound. In chronic wounds, epithelial cells latch onto and pull themselves across the scaffolding of macromolecules of the provisional matrix, such as laminin and fibronectin. Common types of chronic wounds include venous ulcers, pressure injuries/pressure ulcers, diabetic foot ulcers, arterial ulcers, infected open surgical incisions, and contaminated traumatic wounds.

Factors Affecting Wound Healing

Host Factors

Host factors (e.g., age, disease burden, sex, race/ethnicity, compromised immunity, medications, BMI, nutritional status, lifestyle habits including smoking) affect wound healing. Patient characteristics including age are related to the increased incidence and prevalence of chronic wounds. Studies have also shown sex-related differences in wound healing and outcomes.[24]

Aging. Older individuals experience an increased abundance of proinflammatory cytokines and a lower level of growth factors. Unlike relatively healthy patients with acute wounds in which growth factors and cytokines are increased, older individuals with chronic wounds experience decreased levels of growth factors and increased levels of proinflammatory cytokines.[25] However, even in the presence of limited growth factors, patients with chronic wounds experience delayed healing, because their fibroblasts are not as reactive to growth factors' stimuli as those of younger patients.[26–28] Increased activity of MMPs in older adults can also delay healing, degrading collagen and collagen fragments, that are characteristic in older adults. Neutrophil elastase breaks proteins and peptide bonds and tends to be increased in chronic wounds in older patients.[29] Furthermore, degrading proteins (e.g., fibronectin, vitronectin, tenascin) decrease the ECM's reformation and further delay healing.[30]

Comorbidities. Comorbidities may contribute to inflammatory dysfunction in chronic wounds and symptom burden.[31] However, how different comorbidities and other factors affect healing outcomes are virtually unknown in older individuals with chronic wounds.[32] Research has shown that the immune system is affected by host comorbidities that may affect the wound healing process. Some examples of comorbidities have been studied including diabetes, venous insufficiency, congestive heart failure, abnormal white blood cell function, and obesity.[33] Patients who are obese experience low-grade inflammation, which include many components of the traditional

inflammatory response to pathogens, such as local wounding and systemic increases in cytokines.

Additionally, chronic wound healing in older adults can be influenced by comorbidity treatments, medications, antibiotic resistance, additional infected areas, proinflammatory wound condition, systemic inflammation, compromised immunity, poor circulation, poor hydration, and other nutritional deficiencies.[34]

Medications and Antibiotic Resistance. Chemotherapy medications such as antineoplastic agents (e.g., Eloxatin for metastatic colon cancer) can interfere with wound healing by disrupting cellular replication.[35] Meanwhile, other medications, such as antibiotics without cultures and sensitivities that are still prescribed by some practitioners for chronic wounds, may simply be ineffective in treating chronic wounds, especially in patients with antibiotic resistance.[36]

Additional Infections. When an individual has another infection (e.g., viral, bacterial, fungal, protozoal), monocytes can travel to peripheral tissues, including the area of the chronic wound. Monocytes' presence and their eventual differentiation to macrophages can further reinforce inflammation and delay healing.[37]

Proinflammatory Conditions. Proinflammatory conditions, such as vasculitis and rheumatoid arthritis, result in the release of proinflammatory cytokines (e.g., IL-1, IL-6, and TNF-α), which, in turn, promote and prolong the inflammatory process.[38]

C-reactive protein (CRP) is a clinically validated marker of inflammation, and measuring its values can be useful in determining the degree of systemic inflammation, as well as the progress and effectiveness of treatments. Measuring systemic cytokines can also provide much needed understanding of the extent of specific inflammatory proteins in individuals with chronic wounds.

Compromised Immunity. An individual's body's lack of response to wound infection may be due to compromised immunity that may impact wound healing. For example, patients who are HIV positive with AIDS experience compromised immunity, because they have low (<100) CD4+ T-cell counts.[39] Low count in these white blood cells

means limited protection from infection and, thus, prolonged wound healing.[40]

Inadequate Circulation. Patients with circulatory problems (such as in diabetes mellitus and chronic venous ulcers) have impaired tissue repair, because reduced blood flow leads to limited oxygen supply for the wound.[41] Since oxygen is imperative for collagen synthesis by fibroblasts and tissue regeneration, limited blood supply can further delay wound healing.[42]

Poor Nutrition and Hydration. The effects of a patient's nutritional status can be critical to an individual's wound healing. Patients who have frequent diarrhea from medications for their comorbidities (e.g., omeprazole for heartburn or stomach ulcers, NSAIDs for arthritis, metformin for diabetes) can experience nutrient and water deficiencies. Limited water and nutrient supply to the wound can undermine and hinder many of the processes of healing, including epithelialization and collagen synthesis.[43]

Factors suggested to affect wound healing have been summarized in Box 5-7.

Wound Biofilms

Bacterial biofilms are known to contribute to numerous chronic inflammatory diseases, and recent evidence suggests that biofilms also play an important role in impairing healing in chronic skin wounds.[44–48] Wound bacteria that grow in clumps embedded in a thick, self-made, protective, slimy barrier of sugars and proteins are called a wound biofilm. Biofilms are defined as complex, dynamic microbial communities made up of microorganisms (bacteria and fungi) that synthesize and secrete a protective matrix that attaches the biofilm firmly to the surface of the wound bed and also buried deeper under the surface of the wound bed.[48–50] They consist of a single bacterial or fungal species or, more commonly, may be polymicrobial, that is, they contain multiple diverse species that are continuously changing.[54] Biofilms trigger a chronic inflammatory response that results in the accumulation of neutrophils and macrophages surrounding biofilms. The neutrophils and macrophages secrete high levels of ROS that affect the biofilm and the surrounding tissue. Inflammatory cells also secrete high levels of proteases (MMPs and elastase) that can help to break down the attachments between biofilms and the tissue, dislodging the biofilms from the tissue.[58] However, chronically elevated levels of proteases and ROS can also damage normal surrounding tissue, proteins, immune cells, and tissue cells, impairing healing, as is the case in most chronic wound beds.

Predisposing Factors for Development of Wound Biofilms

In vulnerable tissue, biofilms arise from planktonic bacteria attaching and forming a protective community before they are killed by the patient's immune system, by antibiotics, or by debridement. Thus, general conditions that impair the immune system or reduce the effectiveness of antibiotic drugs favor the development of biofilms in wounds. These conditions include ischemia or necrosis of tissue; poor patient nutrition; comorbidities that impair immune function, such as HIV, diabetes, major trauma, and radiation treatment; or treatment with immune-suppressing drugs.

Assessment of Biofilms

The consensus guidelines for identification and treatment of biofilms in chronic and nonhealing wounds[59] states that “biofilms are present in most chronic wounds and are likely to be located both on the surface and in deeper wound layers, but may not be present uniformly across or within the wound” (p. 745).[59] Key consensus statements also confer that “wounds that contain biofilms may not be identified, resulting in ineffective treatment and delayed healing” (p. 745).[59] In chronic wounds, it can be difficult to distinguish biofilms from slough. “Wound biofilms are difficult to visualize macroscopically and slough, debris, and exudate may be visually mistaken for biofilm” (p. 745).[59] Wound slough has been described as a viscous, yellow, and relatively opaque layer on wound beds, while biofilm found in wounds can appear more gel-like and shiny.[60] There is an important link between biofilms and slough. Biofilms stimulate inflammation, which increases vascular permeability and the production of wound exudate and buildup of fibrin slough.[33] Therefore, slough may indicate that biofilm is present in the wound. Unfortunately, chronic skin wounds are frequently assessed with standard clinical microbiology laboratory assays that are designed to culture single, planktonic bacteria, and they do not adequately measure biofilm bacteria. Currently, the most reliable method to confirm the presence of microbial biofilm is specialized microscopy.[61–65] An analysis using special cultivation techniques of biopsies from chronic wounds found that 60% of the specimens contained biofilm structures in comparison with only 6% of biopsies from acute wounds.[47] A recent meta-analysis of published papers that reported on various aspects of chronic wounds found that on average, about 80% of chronic wounds had strong evidence of bacterial biofilms in samples collected from the chronic wounds.[66]

Management of Biofilms

Antibiotics and antiseptics kill single bacteria very easily, but the biofilm matrix can reduce penetration of most chemically reactive antiseptic molecules from reaching all the bacteria, particularly in the center of the biofilm matrix by chemically reacting with and neutralizing the antiseptic molecule, which has been called the “reaction–diffusion

problem." In addition, some bacteria in mature biofilms become metabolically dormant, which is important because antibiotics typically only kill metabolically active bacteria by interfering with essential bacterial processes like cell wall synthesis, energy production, synthesis of proteins, DNA, or RNA.[67] Therefore, bacteria in biofilms become very tolerant to most antibodies, antibiotics, disinfectants, and phagocytic inflammatory cells. Wound biofilms can be effectively treated by a combination of debridement and/or cleansing to remove the biofilms, followed by application of dressings that block new bacteria from reaching the wound and killing bacteria left in the wound bed. This concept for treatment of chronic wounds that likely have biofilm bacteria has been termed "biofilm-based wound care".[68,69] These treatments can heal wounds, but patients must comply with the treatment plan because biofilms can reform within a couple of days and the wound will not heal.[70] The consensus guidelines for identification and treatment of biofilms in chronic and nonhealing wounds states that "important indicators that a wound is likely to contain a biofilm include recalcitrance to treatment with antibiotics or antiseptics" (p. 745).[59]

An ex vivo porcine skin explant biofilm model that preserves key properties of biofilm attached to skin at different levels of maturity (0 to 3 days) was used to assess the efficacy of commercially available antimicrobial dressings and topical treatments.[71] Five types of antimicrobial agents (iodine, silver, polyhexamethylene biguanide, honey, and ethanol) and four types of moisture dressings (cotton gauze, sodium carboxymethylcellulose fiber, calcium alginate fiber, and cadexomer beads) were assessed. Time-release silver gel and cadexomer iodine dressings were the most effective in killing mature biofilm, reducing the level of biofilm-protected bacteria between 10^5 and 10^7 colony-forming units (CFUs) for a total of 10^7 CFUs of biofilm bacterial. In contrast, all other dressing formulations reduced biofilm between $10^{0.3}$ and 10^2 log CFUs in 24 or 72 hours with a single exposure. Similar results were found after *24-hour exposure to silver-release dressings using an in vivo pig burn wound model, demonstrating correlation between the ex vivo* and *in vivo* models. These results indicate that commonly used microbicidal wound dressings vary widely in their ability to kill mature biofilm, and the

efficacy is influenced by time of exposure, number of applications, moisture level, and the formulation of the microbicide, which sustained release formulations providing improved killing.[71] Consensus guidelines for identification and treatment of biofilms in chronic and nonhealing wounds states that “debridement is one of the most important treatment strategies against biofilms, but does not remove all biofilm, and therefore cannot be used alone—this is one of the critical principles of wound bed preparation (tissue, infection/inflammation, moisture balance, and edge of wound) (p. 745).[59] Biofilms can reform rapidly; repeated debridement alone is unlikely to prevent biofilm regrowth, however, effective topical antiseptic application within the time-dependent window can suppress biofilm reformation” (p. 745).[59] These factors have led to an expansion of the original principles of biofilm-based wound care to now include the concept of “step-down then step-up” treatment strategy for chronic wounds.[59] Basically, this strategy emphasizes to initiate treatment of chronic wounds that are suspected to have biofilm with the treatments that are most effective at removing biofilms—especially sharp debridement—combined with the topical and systemic treatments that are most effective at killing and removing biofilm bacteria. As the biofilm bioburden becomes reduced, the level of inflammation will reduce, which will lead to reduction on ROS and proteases in the wound bed. Treatment of the wound bed can then “step-down” to less frequent debridement and to dressings that can effectively kill and reduce planktonic bacteria. As the wound begins to heal, the decision can be made to “step-up” the treatment strategy and use advanced wound treatments such as growth factors, biologically active cell and tissue products, or skin grafts that can dramatically stimulate healing because the wound bed has been properly prepared and these protein-based/cell-based advanced treatments will work well because the levels of ROS and proteases in the wound bed are too low to degrade the advanced treatment products.[59]

Wound Bed Preparation

The **TIME** acronym (**T**issue, **I**nfection/**I**nflammation, **M**oisture balance, and **E**dge of wound) was first developed more than 15 years ago, by an international group of wound healing experts, to provide a framework for a structured approach to wound bed preparation, a basis for optimizing the management of open chronic wounds healing by secondary intention.[72] The framework was therefore termed “wound bed preparation” and was subsequently published in 2003 by Schultz and colleagues.[73] Since then, the TIME acronym has been widely used as a practical guide for the assessment and management of chronic wounds. The clinical observations and interventions relating to wound bed preparation are grouped into four areas, all of which need to be addressed at each wound assessment:

- **T**issue: assessment and debridement of nonviable or foreign material (including host necrotic tissue, adherent dressing material, multiple organism–related biofilm or slough, exudate, and debris) on the surface of the wound
- **I**nfection/Inflammation: assessment of the etiology of each wound, need for topical antiseptic and/or systemic antibiotic use to control infection, and management of inappropriate inflammation unrelated to infection
- **M**oisture imbalance: assessment of the etiology and management of wound exudate
- **E**dge of wound: assessment of nonadvancing or undermined wound edges (and state of the surrounding skin)

However, it should be recognized that the TIME principles are only a part of the systematic and holistic evaluation of each patient at every wound assessment. An article by Leaper et al.[72] examines how new data and evidence generated in the intervening decade impact on the original concepts of TIME and how they are translated into current best practice (Table 5-3).

Table 5-3 Summary Table of Developments Within the TIME Concept

Clinical Observations	WBP	Developments	Factors to Consider
*T*issue	Debridement	**New methods** • Low-frequency noncontact ultrasound (MIST, Sonoca) • Hydrosurgery • Debrisoft wipes **Advances in use of existing methods** • Larvae • Autolytic (honey, hydrogels) • Use of enzymes (collagenase) • Sharp/surgical (new guidelines) • Chemical (antiseptics, i.e., silver, PHMB) **NPWT**—as add-on with existing debridement methods	Use of maintenance debridement Considerations around safe practice • Knowledge • Skills • Competence • Evidence of efficacy
	Wound cleansing	Microbicidal irrigation solutions (Prontosan)	
*I*nfection/ *I*nflammation	Bacterial balance	**Biofilm** • Improved understanding of biofilms and their role in nonhealing wounds • Management—combination strategy to disrupt biofilm and prevent reconstitution (debridement, antiseptic agents) • Detection of biofilm Use of PCR/pyrosequencing techniques to identify bacteria/fungi in wounds	Increased bacterial tolerance to topical/ systemic agents Mixed flora living synergistically Quiescent state of some bacteria in biofilms reduces effectiveness of antibiotics Diagnostic for biofilm detection needed
	Persistent inflammation	Improved understanding of the role of persistent inflammation in chronic/stalled wounds • Role of MMPs and other proteases (diagnostics and inhibitors) • Role of biofilms in promoting wound inflammation	
	Managing infection/ inflammation	• Increased use of antiseptic agents • Role of nanocrystalline silver as an anti-inflammatory • Combination of surfactants with antimicrobials—biofilm disruption • NPWT combined with instillation of microbicidal solutions to reduce levels of planktonic and biofilm bacteria • Alternative use of new or existing agents—e.g., using nanocrystalline silver to dampen down inflammation • Improved healing of wounds treated with custom formulations of topical antibiotics/antiseptics based on bacterial profiles	Diagnostic tests—when and how often? Point-of-care detection Review of appropriate antimicrobials Rotation of products Microbial resistance (particularly to antibiotics)

Clinical Observations	WBP	Developments	Factors to Consider
***M**oisture*	Moisture balance	Improved awareness of need to maintain appropriate moisture levels	Dressing selection—what do we need to consider? • Absorption • Retention • Patient comfort • Bacterial pool Skin sensitivity or allergy
	Exudate	Improved understanding of exudate composition—differences between acute and chronic wound fluid • Damaging proteolytic activity of chronic wound fluid Relationship of exudate with bacterial burden and biofilm formation Selection of appropriate dressings or devices for exudate management (i.e., new super absorbers) Greater emphasis on moisture management **NPWT**—for removal and containment of large exudate volumes	
***E**dge of wound*		Epithelial edge advancement Improved state of surrounding skin **Evaluation**—check whether wound is closing Use of NPWT to encourage contraction Adjunct therapies (EMT, laser, ultrasound, systemic oxygen therapy)	Revisiting existing therapies Alternative use of products, e.g., using NPWT to splint wounds Role of diagnostics/ theranostics

From Gould, L., Abadir, P., Brem, H., et al. "Chronic Wound Repair and Healing in Older Adults: Current Status and Future Research." *Wound Repair and Regeneration* 23(1):1-13, 2015. doi: 10.1111/wrr.12245.

Practice Point

The TIME framework can be used to provide a basis for discussion of wound assessment and treatment goals with the patient.

A summary of their findings for each element of the TIME principles is as follows[72]:

1. **T**issue: A major advance has been the recognition of the value of repetitive and maintenance debridement and wound cleansing, both in time-honored and novel methods (notably using negative pressure wound therapy [NPWT] and hydrosurgery).

2. **I**nfection/Inflammation: Clinical recognition of infection (and noninfective causes of persisting inflammation) is critical. The concept of a bacterial continuum through contamination, colonization, and infection is now widely accepted, together with the understanding of biofilm presence. There has been a return to topical antiseptics to control bioburden in wounds, emphasized by the awareness of increasing antibiotic resistance. Recognition of the importance of biofilms (and the need for a simple diagnostic), use of NPWT, evolution of topical antiseptic therapy as dressings and for wound lavage (notably, silver and polyhexamethylene biguanide), and expanded insight of the role of molecular biological processes in chronic wounds (with emerging diagnostics and theranostics).
3. **M**oisture: The relevance of excessive or insufficient wound exudate and its molecular components has led to the development and use of a wide range of dressings to regulate moisture balance, to protect periwound skin, and to optimize healing.
4. **E**dge of wound: Several treatment modalities are being investigated and introduced to improve epithelial advancement, which can be regarded as the clearest sign of wound healing.

Practice Point

In addition to the TIME assessment of the wound, there should be an evaluation of the surrounding skin as this can provide valuable information to determine whether the wound is progressing or deteriorating.

Symptoms and Symptom Clusters Associated with Chronic Wounds

The communication between the brain and peripheral immunity is essential to regulating stress and altering behaviors.[51] Some examples demonstrate that immune molecules such as cytokines induced by T cells directly affect pain,[52] cognition,[53] and sickness behaviors. Sickness behaviors such as pain and fatigue, depression, and anxiety are common in people who are undergoing illness.[51,52] People with chronic wounds experience both psychological distress and sickness behaviors.[54–57]

Symptoms frequently accompany individuals with chronic wounds.[74,75] Many patients experience four or more concurrent symptoms, which frequently include pain, fatigue, anxiety, depression, and sleep disturbances.

Pain

Pain commonly affects many individuals with chronic wounds and greatly complicates their ability to care for their wound and perform self-care.[76] Pain may result in delay of treatment and reduce quality of life.[74,75,77–80] Furthermore, pain associated with chronic wounds is often not adequately identified or treated, which may result in delay in wound healing.[74,81,82]

Cognitive Changes, Fatigue, Depression, and Anxiety

Many individuals with chronic wounds also experience cognitive changes, fatigue, depression, and anxiety that may also occur with pain.[75] Chronic pain, anxiety, and depressive symptoms may also impair cognitive abilities.[81] There is increasing evidence that individuals with chronic wounds experience both event-related pain and somatic pain.[82] Depression has also been associated with delayed wound healing.[83,84] Though pain and cognitive/affective symptoms contribute to outcomes in chronic wounds, there is a paucity of research that focuses on symptoms in individuals with chronic wounds, which requires future study.

Summary

The molecular and cellular environment of chronic wounds differs substantially from that of acute healing wounds. Specifically, nonhealing wounds have chronically elevated proinflammatory cytokines, which lead to chronically elevated levels of proteases (MMPs and neutrophil elastase) and ROS that degrade the components that are essential to healing, such as ECM components, growth factors, and receptors. Wound bed preparation is an overarching concept, applied in clinical practice using the TIME framework, which can help clinicians to determine whether a wound is progressing or deteriorating.

A number of host factors including age, disease burden, sex, race/ethnicity, compromised immunity, medications, BMI, nutritional status, and lifestyle habits including smoking have been shown to affect wound healing. Bacterial biofilms also play an important role in impairing healing in chronic skin wounds. There appear to be common symptoms/symptom clusters associated with wounds, which frequently include pain, fatigue, anxiety, depression, and sleep disturbances, which need to be addressed as part of the comprehensive management of the patient.

PATIENT SCENARIO

Clinical Data

Mrs. A is a 72-year-old woman who collapsed at home, catching her right shin on a coffee table. She was taken to hospital for investigation of the cause of her fall. She had extensive bruising and a hematoma on her right lower leg. On further investigation, she was found to have a lower respiratory tract infection, and she was admitted for treatment of this. Mrs. A is a smoker and has chronic obstructive pulmonary disease (COPD) and ischemic heart disease. She is on dual antiplatelet therapy following a myocardial infarction and coronary artery stents earlier in the year. She frequently takes oral steroids for infective exacerbations of her COPD.

Case Discussion

Mrs. A's leg was initially managed conservatively because of her chest infection; however, 5 days later, it became increasingly painful, and the skin around the hematoma was erythematous and hot. She was referred to the trauma surgeons, who diagnosed infection of the hematoma, and elected to evacuate it, creating an open wound on her leg (Fig. 5-2A). At this stage, there is an acute surgical wound, but one that would be classified as Class IV (dirty/infected) according to the Centers for Disease Control classification.[85]

Figure 5-2. (A) Hematoma post evacuation. **(B)** Post treatment with topical antimicrobial dressings.

Following her procedure, negative pressure wound therapy was applied to her leg. Microbiology samples sent from the operating room were used to guide antibiotic therapy, but once the infected hematoma had been drained, the erythema settled quickly. Multidisciplinary input was required to make a plan going forward for Mrs. A's management.[86] In order to make a plan for her wound management, her other medical problems, as well as the underlying causes of the wound, were considered.[86] Split skin grafting (SSG) was not carried out at her initial operation because of the infection present. While it was felt that a successful SSG would decrease the time taken for the wound to heal, there were factors present that increased the risk of graft failure. Mrs. A did not feel she would be able to stop smoking, and she did not want to have a second wound on her leg. Mrs. A's skin quality had been affected by the multiple courses of steroids she had taken for her lung disease. Additionally,

the risks of stopping Mrs. A's anticoagulant medications were thought to outweigh the benefits, and this increased the risk of bleeding at the donor site. It was therefore decided that SSG would not be attempted and the tissue viability nurses/WOC nurse took the lead in the care of Mrs. A's leg.

The wound initially progressed well with the NPWT; however, there was evidence of damage in the fragile periwound skin, despite the use of barrier creams. NPWT was therefore discontinued, and a Hydrofiber primary dressing was applied to manage the exudate. At each visit, the nurses measured the wound to assess progress. The wound became static, with no reduction in size over 5 weeks. On examination at this time, the wound bed was covered with a gelatinous slough. While there were no signs of spreading infection (cellulitis, malaise), the wound edge appeared inflamed. By this point, the wound would be classified as chronic, because of its duration, and the lack of progression through the stages of wound healing as expected. The measurements also indicated it had become a nonhealing wound. It was thought that the bioburden in the wound may be arresting the normal healing process. Debridement of the wound bed was therefore undertaken. Topical local anesthetic cream was used to enable sharp debridement with a ring curette. A polyhexamethylene biguanide–containing wound cleanser was also used at dressing changes between debridement procedures. An absorbent antimicrobial dressing (silver-containing Hydrofiber) was applied. The wound began to make progress again, with evidence of neoepithelialization at the wound edge and contraction (Fig. 5-2B). The antimicrobial dressings were discontinued, and the wound continued to make progress.

Show What You Know

1. **Which of the following would be considered abnormal in the inflammatory phase of wound healing?**
    A. Redness
    B. Bleeding
    C. Pain
    D. Swelling
    E. Heat

2. **Wound biofilm can be effectively treated by:**
    A. debridement.
    B. cleansing.
    C. dressings to block new bacteria and kill existing bacteria.
    D. antibiotics.
    E. a combination of debridement, cleansing, and dressings.

3. **Chronic wounds and comorbidities are associated with systemic inflammation, and evidence indicates that the clinician should assess for:**
    A. sickness behaviors that may include pain, fatigue, depression, sleeping difficulties, and cognitive changes.
    B. symptom clusters that have been identified in individuals with chronic wounds.
    C. pain that commonly affects many individuals with chronic wounds and ability to care for their wound and perform self-care.
    D. all of the above.

Answers to these questions are found on page 682.

References

1. Demidova-Rice, T.N., Hamblin, M.R., Herman, I.M. “Acute and Impaired Wound Healing: Pathophysiology and Current Methods for Drug Delivery, Part 1: Normal and Chronic Wounds: Biology, Causes, and Approaches to Care.” Advances in Skin and Wound Care 25(7):304-14, July 2012.
2. Baum, C.L., Arpey, C.J. “Normal Cutaneous Wound Healing: Clinical Correlation with Cellular and Molecular Events.” Dermatologic Surgery 31:674-86, June 2005.
3. Schultz, G.S., Davidson, J.M., Kirsner, R.S., et al. “Dynamic Reciprocity in the Wound Microenvironment.” Wound Repair and Regeneration 19:134-48, 2010.
4. Gill, G.E., Parks, W.C. “Metalloproteinases and Their Inhibitors: Regulators of Wound Healing.” The International Journal of Biochemistry & Cell Biology 40:1334-47, 2008.
5. Midwood, K.S., Williams, L.V., Schwarzbauer, J.E. “Tissue Repair and the Dynamics of the Extracellular Matrix.” The International Journal of Biochemistry & Cell Biology 36:1031-7, 2004.
6. McAnulty, R.J. “Fibroblasts and Myofibroblasts: Their source, Function and Role in Disease.” The International Journal of Biochemistry & Cell Biology 39:666-71, 2007.
7. Li, J., Chen, J., Kirsner, R. “Pathophysiology of Acute Wound Healing.” Clinics in Dermatology 25:9-18, 2007.
8. Venus, M., Waterman, J., McNab, I. “Basic Physiology of the Skin,” Surgery 28(10):469-72, 2010.
9. Li, J., Zhang, Y.P., Kirsner, R.S. “Angiogenesis in Wound Repair: Angiogenic Growth Factors and the Extracellular Matrix.” Microscopy Research and Technique 60:107-14, 2003.
10. Diegelmann, R.F., Evans, M.C. “Wound Healing: An Overview of Acute, Fibrotic and Delayed Healing.” Frontiers in Bioscience 9:283-9, January 2004.
11. Stavrou, D., Haik, J., Weissman, O., et al. “From Traction and Contraction to Wound Closure.” Journal of Wound, Ostomy & Continence Nursing 36(4):365-6, July/August 2009.
12. Desmoulière, A., Chaponnier, C., Gabbiana, G. “Tissue Repair, Contraction, and the Myofibroblast.” Wound Repair and Regeneration 13:7-12, 2005.
13. Bates-Jensen, B.M., Woolfolk, N. “Acute Surgical Wound Management.” In C. Sussman, B.M. Bates-Jensen, eds. Wound Care: A Collaborative Practice Manual for Health Professionals. Baltimore, MD: Lippincott Williams and Wilkins, 2007:322-35.
14. Sussman, C., Bates-Jensen, B.M. “Wound Healing Physiology: Acute and Chronic.” In C. Sussman, B.M. Bates-Jensen, eds. Wound Care: A Collaborative Practice Manual for Health Professionals. Baltimore, MD: Lippincott Williams and Wilkins, 2007:21-51.

15. Lazarus, G.S., et al. “Definitions and Guidelines for Assessment for Wounds and Evaluation of Healing.” Archives of Dermatology 130(4):489-93, April 1994.
16. Fletcher, J. “Differences between Acute and Chronic Wounds and the Role of Wound Bed Preparation.” Nursing Standard 22(24):62-8, 2008. Available at: http://rcnpublishing.com/doi/abs/10.7748/ns2008. 02.22.24.62.c6412.
17. NHS. “Treatment: Venous Leg Ulcer.” 2019. Available at: https://www.nhs.uk/conditions/leg-ulcer/treatment/
18. Guo, S., DiPietro, L.A. “Factors Affecting Wound Healing.” Journal Dental Research 89(3):219-29, 2010.
19. Doughty, D. “Why Won’t Some Wounds Heal.” Advances in Skin & Wound Care 17(7):342-4, September 2004.
20. Widgerow, A.D. “Deconstructing the Stalled Wound.” Wounds 24(3):58-66, March 2012.
21. Phillips, T.J., et al. “Effect of Chronic Wound Fluid on Fibroblasts.” Journal of Wound Care 7(10):527-32, November 1998.
29. Hart, J. “Inflammation 2: Its Role in the Healing of Chronic Wounds.” Journal of Wound Care 11:245-9, July 2002.
30. Yager, D.R., Nwomeh, B.C. “The Proteolytic Environment of Chronic Wounds.” Wound Repair and Regeneration 7(6):433-41, November-December 1999.
31. Ligi, D., Mosti, G., Croce, L., et al. “Chronic Venous Disease—Part I: Inflammatory Biomarkers in Wound Healing.” Biochimica et Biophysica Acta (BBA)-Molecular Basis of Disease 1862(10):1964-74, 2016.
32. Barrientos, S., Stojadinovic, O., Golinko, M.S., et al. “Growth Factors and Cytokines in Wound Healing.” Wound Repair and Regeneration 16(5):585-601, 2008.
33. Ågren, M.S., Steenfos, H.H., Dabelsteen, S., et al. “Proliferation and Mitogenic Response to PDGF-BB of Fibroblasts Isolated from Chronic Venous Leg Ulcers is Ulcer-Age Dependent.” Journal of Investigative Dermatology 112(4):463-9, 1999.
34. Falanga, V., Zhou, L., Yufit, T. “Low Oxygen Tension Stimulates Collagen Synthesis and COL1A1 Transcription through the Action of TGF-β1.” Journal of Cellular Physiology 191(1), 42-50, 2002.
35. Kim, B.C., Kim, H.T., Park, S.H., et al. “Fibroblasts from Chronic Wounds Show Altered TGF-β-Signaling and Decreased TGF-β Type II Receptor Expression.” Journal of Cellular Physiology 195(3):331-6, 2003.
36. Enoch, S., Price, P. “Cellular, Molecular and Biochemical Differences in the Pathophysiology of Healing between Acute Wounds, Chronic Wounds and Wounds in the Aged.” World Wide Wounds 13:1-17, 2004.
37. Ashcroft, G.S., Greenwell-Wild, T., Horan, M.A., et al. “Topical Estrogen Accelerates Cutaneous Wound Healing in Aged Humans Associated with an Altered Inflammatory Response.” The American Journal of Pathology 155(4):1137-46, 1999.

38. Sarkar, D., Fisher, P.B. “Molecular Mechanisms of Aging-Associated Inflammation.” Cancer Letters 236(1):13-23, 2006.
39. Gould, L., Abadir, P., Brem, H., et al. “Chronic Wound Repair and Healing in Older Adults: Current Status and Future Research.” Wound Repair and Regeneration 23(1):1-13, 2015. doi: 10.1111/wrr.12245.
40. Wolcott, R.D., Rhoads, D.D., Dowd, S.E. “Biofilms and Chronic Wound Inflammation.” Journal of Wound Care 17(8):333-41, 2008.
41. Moreo, K. “Understanding and Overcoming the Challenges of Effective Case Management for Patients with Chronic Wounds.” The Case Manager 16(2):62-7, 2005.
42. Pollack, S.V. “Wound Healing: A Review. IV. Systemic Medications Affecting Wound Healing.” The Journal of Dermatologic Surgery and Oncology 8(8):667-72, 1982.
43. Howell-Jones, R., Wilson, M., Hill, K., et al. “A Review of the Microbiology, Antibiotic Usage and Resistance in Chronic Skin Wounds.” Journal of Antimicrobial Chemotherapy 55(2):143-9, 2005.
44. Shi, C., Pamer, E.G. “Monocyte Recruitment during Infection and Inflammation.” Nature Reviews Immunology 11(11):762, 2011.
45. Choy, E.H., Panayi, G.S. “Cytokine Pathways and Joint Inflammation in Rheumatoid Arthritis.” New England Journal of Medicine 344(12):907-16, 2001.
46. Njunda, A.L., Nsagha, D.S., Assob, J.C., et al. “In vitro Antifungal Susceptibility Patterns of *Candida albicans* from HIV and AIDS Patients Attending the Nylon Health District Hospital in Douala, Cameroon.” *Journal of Public Health in Africa* 3(1):e2, 2012.
47. Weledji, E., Kamga, H., Assob, J., et al. “A Critical Review on HIV/AIDS and Wound Care.” African Journal of Clinical and Experimental Microbiology 13(2):66-73, 2012.
48. Greenhalgh, D.G. “Wound Healing and Diabetes Mellitus.” Clinics in Plastic Surgery, 30(1):37-45, 2003.
49. Trabold, O., Wagner, S., Wicke, C., et al. “Lactate and Oxygen Constitute a Fundamental Regulatory Mechanism in Wound Healing.” Wound Repair and Regeneration 11(6):504-9, 2003.
50. Stechmiller, J.K. “Understanding the Role of Nutrition and Wound Healing.” Nutrition in Clinical Practice 25(1):61-8, 2010.
51. Costerton, J.W., Stewart, P.S., Greenberg, E.P. “Bacterial Biofilms: A Common Cause of Persistent Infections.” Science 284(5418):1318-22, 1999.
52. Costerton, J.W. “The Etiology and Persistence of Cryptic Bacterial Infections: A Hypothesis.” Review of Infectious Diseases 6(Suppl 3):S608-16, 1984.
53. Hall-Stoodley, L., Stoodley, P. “Evolving Concepts in Biofilm Infections.” Cell Microbiology 11(7):1034-43, 2009.
54. Wolcott, R.D., Rhoads, D.D., Bennett, M.E., et al. “Chronic Wounds and the Medical Biofilm Paradigm.” Journal of Wound Care 19(2):45-50, 52, 2010.

55. Stoodley, P., Sauer, K., Davies, D.G., et al. “Biofilms as Complex Differentiated Communities.” Annual Review of Microbiology 56:187-209, 2002.
56. Fazli, M., Bjarnsholt, T., Kirketerp-Moller, K., et al. “Non-Random Distribution of *Pseudomonas aeruginosa* and *Staphylococcus aureus* in Chronic Wounds.” Journal of Clinical Microbiology 47(12):4084-9, October 2009.
57. Kirketerp-Moller, K., Jensen, P.O., Fazli, M., et al. “Distribution, Organization, and Ecology of Bacteria in Chronic Wounds.” Journal of Clinical Microbiology 46(8):2717-22, 2008.
22. Kipnis, J. “Immune System: The “Seventh Sense”.” Journal of Experimental Medicine 215(2):397-8, 2018.
23. Scholz, J., Woolf, C.J. “The Neuropathic Pain Triad: Neurons, Immune Cells and Glia.” Nature Neuroscience 10(11):1361, 2007.
24. Kipnis, J., Derecki, N.C., Yang, C., et al. “Immunity and Cognition: What Do Age-Related Dementia, HIV-Dementia and ‘Chemo-Brain’ Have in Common?” Trends in Immunology 29(10):455-63, 2008.
25. McCusker, R.H., Kelley, K.W. “Immune–Neural Connections: How the Immune System’s Response to Infectious Agents Influences Behavior.” Journal of Experimental Biology 216(1):84-98, 2013.
26. Louveau, A., Harris, T.H., Kipnis, J. “Revisiting the Mechanisms of CNS Immune Privilege.” Trends in Immunology 36(10):569-77, 2015.
27. Louveau, A., Smirnov, I., Keyes, T.J., et al. “Structural and Functional Features of Central Nervous System Lymphatic Vessels.” Nature 523(7560):337, 2015.
28. Fung, T.C., Olson, C.A., Hsiao, E.Y. “Interactions between the Microbiota, Immune and Nervous Systems in Health and Disease.” Nature Neuroscience 20(2):145-55, 2017.
58. Gibson, D., Cullen, B., Legerstee, R., et al. “MMPs Made Easy.” Wounds International 1(1):1-6, 2010.
59. Schultz G, Bjarnsholt, T., James, G.A., Leaper, D.J., et al. “Consensus Guidelines for the Identification and Treatment of Biofilms in Chronic Non-Healing Wounds.” Wound Repair and Regeneration 25:744-57, 2017.
60. Hurlow, J., Bowler, P.G. “Clinical Experience with Wound Biofilm and Management: A Case Series.” Ostomy Wound Management 55(4):38-49, 2009.
61. Dowd, S.E., Sun, Y., Secor, P.R., et al. “Survey of Bacterial Diversity in Chronic Wounds Using Pyrosequencing, DGGE, and Full Ribosome Shotgun Sequencing.” Bio Med Central Microbiology 8(1):43, 2008.
62. Edwards, R., Harding, K.G. “Bacteria and Wound Healing.” Current Opinion in Infectious Diseases 17(2): 91-6, 2004.
63. Costerton, W., Veeh, R., Shirtliff, M., et al. “The Application of Biofilm Science to the Study and Control of Chronic Bacterial Infections.” Journal of Clinical Investigation 112(10):1466-77, 2003.
64. Kaeberlein, T., Lewis, K., Epstein, S.S. “Isolating ‘Uncultivable’ Microorganisms in Pure Culture in a Simulated Natural Environment.” Science 296(5570): 1127-9, 2002.

65. Bjarnsholt, T., Kirketerp-Moller, K., Jensen, P.O., et al. “Why Chronic Wounds Will Not Heal: A Novel Hypothesis.” Wound Repair and Regeneration 16(1): 2-10, 2008.
66. Malone, M., Bjarnsholt, T., McBain, A.J., et al. “The Prevalence of Biofilms in Chronic Wounds: A Systematic Review and Meta-Analysis of Published Data.” Journal of Wound Care 26(1):20-5, 2017.
67. Hoiby, N., Bjarnsholt, T., Givskov, M., et al. “Antibiotic Resistance of Bacterial Biofilms.” International Journal of Antimicrobial Agents 35(4):322-32, 2010.
68. Wolcott, R.D., Rhoads, D.D. “A Study of Biofilm-Based Wound Management in Subjects with Critical Limb Ischaemia.” Journal of Wound Care 17(4):145-2, 154, 2008.
69. Wolcott, R.D., Dowd, S., Kennedy, J., et al. “Biofilm-Based Wound Care.” Advances Wound Care 1:311-7, 2010.
70. Wolcott, R.D., Rumbaugh, K.P., James, G., et al. “Biofilm Maturity Studies Indicate Sharp Debridement Opens a Time-Dependent Therapeutic Window.” Journal of Wound Care 19(8):320-8, 2010.
71. Phillips, P.L., Yang, Q.P., Davis, S., et al. “Antimicrobial Dressing Efficacy against Mature *Pseudomonas aeruginosa* Biofilm on Porcine Skin Explants.” International Wound Journal 12(4):469-83, 2015, doi: 10.1111/iwj.12142.
72. Leaper, D.J., Schultz, G., Carville, K., et al. “Extending the TIME Concept: What Have We Learned in the Past 10 Years?” International Wound Journal 9(Suppl. 2):1-19, 2012.
73. Schultz, G.S. “Wound Bed Preparation. A Systemic Approach to Wound Bed Management.” Wound Repair and Regeneration 11(Suppl):S1-28, 2003.
74. Edwards, H., Finlayson, K., Skerman, H., et al. “Identification of Symptom Clusters in Patients with Chronic Venous Leg Ulcers.” Journal of Pain and Symptom Management 47(5):867-75, 2014.
75. Kelechi, T.J., Mueller, M., Dooley, M. “Sex Differences in Symptom Severity and Clusters in Patients with Stage C4 and Stage C5 Chronic Venous Disease.” European Journal of Cardiovascular Nursing 16(1), 28-36, 2017.
76. Alföldi, P., Wiklund, T., Gerdle, B. “Comorbid Insomnia in Patients with Chronic Pain: A Study Based on the Swedish Quality Registry for Pain Rehabilitation (SQRP).” Disability and Rehabilitation 36(20), 1661-9, 2014.
77. Althaus, A., Hinrichs-Rocker, A., Chapman, R., et al. “Development of a Risk Index for the Prediction of Chronic Post-Surgical Pain.” European Journal of Pain 16(6):901-10, 2012.
78. Haythornthwaite, J.A., Menefee, L.A., Heinberg, L.J., et al. “Pain Coping Strategies Predict Perceived Control Over Pain.” Pain 77(1):33-9, 1998.
79. Poobalan, A.S., Bruce, J., Smith, W.C.S., et al. “A Review of Chronic Pain after Inguinal Herniorrhaphy.” The Clinical Journal of Pain 19(1):48-54, 2003.
80. Saxe, J.M., Smith, V., McNerney, K. “A Blueprint to Managing Multiple Chronic Conditions and Pain.” The Journal of Family Practice 62(12 Suppl 1):S4-11, 2013.

81. Seminowicz, D.A., Davis, K.D. “A Re-Examination of Pain-Cognition Interactions: Implications for Neuroimaging.” Pain 130(1-2):8-13, 2007.
82. Rosique, R.G., Rosique, M.J., Farina Junior, J.A. “Curbing Inflammation in Skin Wound Healing: A Review.” International Journal of Inflammation 2015:316235, August 2015. doi: 10.1155/2015/316235.
83. Cole-King, A., Harding, K.G. “Psychological Factors and Delayed Healing in Chronic Wounds.” Psychosomatic Medicine 63(2):216-20, 2001.
84. Kiecolt-Glaser, J.K., Glaser, R. “Depression and Immune Function: Central Pathways to Morbidity and Mortality.” Journal of Psychosomatic Research, 53(4):873-6, 2002. doi: https://doi.org/10.1016/S0022-3999(02)00309-4.
85. Simmons, B.P. “Guideline for Prevention of Surgical Wound Infections.” Infection Control 3(2):187-96, 1982.
86. Vowden, P. “Hard-to-Heal Wounds Made Easy.” Wounds International 2(4):1-6, November 2011.

6

Wound Assessment

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Objectives

After completing this chapter, you'll be able to:

- state reasons for performing a wound assessment
- list the parameters of a complete wound assessment
- differentiate between partial- and full-thickness injury
- describe useful photographic techniques for wound documentation
- discuss wound documentation using an electronic medical record and electronic health record.

The Wound

Reliable, consistent, comprehensive, and accurate wound (ulcer) description and documentation are essential components of a wound assessment. Wound assessment allows classification of the wound by etiology as well as by its appearance and characteristics. The etiology will help guide the management of the patient from an overall medical perspective (e.g., classification as an arterial wound mandates an aggressive interrogation of the patient's vascular system), while the appearance and characteristics will provide a basis for local and topical wound management (e.g., the necrotic wound with adequate blood supply and consist with patient care goals will include debridement). Not only does it provide objective data to confirm wound progress, but it can also serve to alert clinicians about wound deterioration.[1] Wound description and documentation also enhance communication among healthcare providers, patients, and care settings.[1,2] Assessment of wounds is important because several clinical characteristics, such as new or increasing pain, new or increasing cellulitis, new or increasing purulent or nonpurulent drainage, and significant undermining, have been reported to constitute a wound emergency.[2] Appropriate assessment and monitoring of wound healing are founded on scientific principles.[3] For the purposes of simplicity, the term "wound" will be used throughout this chapter. The reader should understand that the assessment terms and technologies discussed in this chapter apply to both wounds and ulcers.

The management of acute and chronic wounds has progressed into a highly focused area of practice, with physicians, nurses, therapists, and other professionals expanding their practice in this challenging arena. Care plans, treatment interventions, case management, and discharge planning, as well as ongoing patient and wound management, are all based on the initial and subsequent wound assessments. The total patient assessment, inclusive of any comorbid conditions and lifestyle, must also be a part of any comprehensive wound assessment. This chapter addresses the key assessment parameters of a patient with a wound admitted to any

healthcare setting, including the importance of a history and physical examination, how to assess a wound, essential practice points, and examples of accurate and thorough documentation tools.

Wound Definitions

A wound is a disruption of normal anatomic structure and function.[4] Wounds are classified as either acute or chronic. Acute wounds can result from trauma or surgery. According to Larazus and colleagues,[4] acute wounds proceed through an orderly and timely healing process with the eventual return of anatomic and functional integrity. Chronic wounds, on the other hand, fail to proceed through this process and lose the cascade effect of wound healing and sustained anatomic and functional integrity. Stated simply, wounds may be classified as those that repair themselves or can be repaired in an orderly and timely process (acute wounds) and those that don't (chronic wounds).[4] See Chapter 5 for a more detailed description of wound healing. In the United States, the current Centers for Medicare and Medicaid Services (CMS) definition of a chronic wound includes a time frame of greater than 30 days' duration for complete healing.[5]

Wound Etiology and Classification

The etiology or cause of a wound must be determined before appropriate interventions can be implemented. This is especially important as many wounds can have mixed etiologies. Ensuring a differential diagnosis is correct is not always easy, but learning the typical characteristics of a wound type can be helpful. The majority of wounds can be classified as surgical, traumatic, neuropathic, vascular, or pressure-related etiology. Others can result from a variety of reasons and are classified as having an atypical etiology. For example, an acute wound caused by a bite (animal, insect, spider, or human) requires a different care plan than a wound caused by a burn. A patient who has an animal bite may require additional testing to rule out damage to nerves, tendons, ligaments, or bones, as well as determination of rabies or rabies vaccination status of the animal and the need for tetanus immunization.[6] The pathologic etiology will provide the basis for additional testing and evaluation to start the wound assessment process and basis for correct classification (see Practice Point: The Nine Cs of Wound Assessment).

Practice Point

The Nine Cs of Wound Assessment

Wound assessment is needed for the following nine reasons:

- Cause(s) of the wound
- Clear picture of what the wound looks like
- Comprehensive picture of the patient
- Contributing factors
- Components of the wound care plan
- Communication to other healthcare providers
- Continuity of care
- Centralized location for wound care information

- Complications from the wound

Initial Patient Assessment

Obtain a thorough history and a complete physical examination on every patient admitted into your care. Obtaining a patient history provides information on relevant disease processes, comorbidities, medications the patient is taking, and family history of conditions that can impact the etiology and potentially the treatment and healing of the wound. In addition, the patient history may reveal information that explains previous wound healing concerns, infection, nutritional status, and other core information needed to develop the individualized plan of care. A detailed patient medical and social history should direct additional questioning on any abnormal lab findings as well as a history of diabetes, vascular conditions, or an immune-compromised state. Assess pain and the patient/family knowledge regarding wounds. Therapies received as part of a prior health condition, such as radiation at the site of a wound, as well as history of a pressure injury increases risk to that area, are also important factors that can contribute to impaired healing and delay appropriate management strategies[1] (see “Radiation Wounds” in Chapter 26, Palliative Skin and Wound Care). Use the assessment data to determine whether the wound is healable, maintenance, or nonhealable.[7] See Table 6-1.

Table 6-1 Healability of a Wound

"Evaluate the Person's Ability to Heal"	Definition
Healable	"The wound has several attributes including an adequate blood supply; the cause of the wound must be corrected; existing cofactors, conditions, or medications that could potentially delay healing must be optimized or ideally corrected."
Maintenance	"is a wound that may be healable but that either the healthcare system factors or patient-related issues are preventing the wound from healing."
Nonhealable	"is a wound that does not have adequate blood supply to support healing or the cause cannot be corrected."

Source: Sibbald, R.G., Goodman, L., Woo, K.Y., et al. "Special Considerations in Wound Bed Preparation 2011: An Update ©." *Advances in Skin & Wound Care* 24(9):415-36, quiz 436-8, 2011.

Family support and patient and family functional abilities should be evaluated as well. Involving other services and/or departments (e.g., social work, case management, pastoral care) early in the care

planning process is crucial to developing a comprehensive plan of care for the wound patient. Case managers can be invaluable in determining the continuity of care across healthcare settings by asking the following questions during the initial assessment:

- What are the patient's values and goals of care?
- Can the patient care for himself or herself?
- Is there a caregiver available to assist with care after discharge?
- Can the patient change his or her own dressings?
- Who will put on and help remove compression stockings?
- Can the patient afford to purchase the necessary wound products/items?
- Does the patient/family know how to care for the stockings or other equipment?

Asking these questions is vital to conducting a comprehensive assessment of a patient with a wound.

Physical Examination

The first thing to do before addressing any wound is to perform an overall physical assessment of the patient. A useful acronym to guide this process is HEIDI[8] (Box 6-1).

Evaluation of the skin, including any skinfolds, pressure points, healed pressure injury sites, old scars or lesions, indications of previous surgeries, and the presence of vascular, neuropathic, or pressure injuries, should be noted. The appearance of the skin, nails, and hair on the extremities should be assessed. Appraisal of skin color, temperature, capillary refill, pulses, and edema are also important elements of a thorough physical examination (see Chapter 4, Skin: An Essential Organ).

Box 6-1 HEIDI[8]

- **H**istory
  - The patient's medical, surgical, pharmacological, and social history
- **E**xamination
  - Of the patient as a whole; then focus on the wound
- **I**nvestigation
  - What bloods, x-rays, or scans do you require to help make your diagnosis?
- **D**iagnosis
- **I**mplementation
  - Of the plan of care

Different types of wounds require different considerations. Dehisced surgical wounds may have opened due to an infection or may heal poorly due to underlying disease processes, current medications (such as steroids), or malnutrition. Hemosiderin staining (reddish-brown color), caused by the chronic leakage of red blood cells into the soft tissue of the lower leg, is a classic sign of venous hypertension

and often seen in a person with a venous insufficiency ulcer (Fig. 6-1). If not managed with compression, this leakage often leads to venous ulcers. Arterial ulcers often present with the classic signs of hair loss, weak or absent pulse, and very thin, shiny, taut skin. Neuropathic ulcers require intense evaluation to determine the extent of the neuropathy. Patients with diabetes are prone to callus formations and pressure points even when off-loading interventions are in place. Both are easily noted on examination.

Figure 6-1. Hemosiderin deposit.

In persons with darkly pigmented skin, early detection of ulceration that relies only on visual inspection by the clinician to note erythema and color changes (such as a stage 1 pressure injury) remains a clinical challenge. The lack of a tool to help clinicians detect erythema

in darkly pigmented skin hampers early detection of tissue injury. In a study in which 28 of 56 subjects had darkly pigmented skin, the authors showed that use of multispectral images of the ulcers resulted in algorithms that enhanced detection of erythema in darkly pigmented skin.[9] Assess for differences in skin temperature at the opposite site, skin tenderness, changes in tissue consistency, and pain.[3]

A comprehensive patient examination will reveal areas of concern for wound development and can pinpoint wound origins as well as why healing is not progressing in some wounds. Based on the comprehensive assessment and the determination as to whether a wound is healable, maintenance, or nonhealable, appropriate goals and treatment plans can be developed[7,10] (see Table 6-1). Developing realistic goals and care plans, performing regular follow-up examinations, and ensuring patient adherence to the plan of care are all key markers for successful outcomes.

Wound Assessment and Classification

Wound assessment—a written record and picture of the current status and progress of a wound—is a cumulative process of observation, data collection, and evaluation at each encounter. As such, it's an important component of patient care. A wound assessment includes a record of your initial assessment, ongoing changes in the wound bed and periwound area, and treatment interventions. The initial assessment serves as the baseline for future comparisons, with ongoing assessments occurring at least weekly and when significant changes occur throughout the healing process.[3]

Practice Point

Because a wound can change rapidly, it is important to assess wounds for changes that could signal the need to modify treatment.[3]

Although the frequency of wound assessment is often determined by individual agency or institutional guidelines, treatment modalities, regulatory guidelines, and wound characteristics also play a role in determining assessment frequency.[11] According to international guidelines, pressure injuries should be evaluated at a minimum on admission, weekly, with any change in the patient's activity or functional ability, and with any signs of deterioration of their condition.[3] Frequency of assessment is also determined by the wound severity, the patient's overall condition, the patient's environment, and the goals and plans of care.[11] Acute care patients often receive wound assessments daily or with each dressing change. In long-term care facilities, pressure injuries must be assessed on admission, with each dressing change, and at least weekly,[12,13] and

more often when indicated by wound complications or changes in wound characteristics, an evaluation of the PU/PI should be documented.[13] Home care assessments are usually based on the frequency of the home visits but often occur weekly and/or with each licensed nurse visit. Regardless of the setting, however, the frequency of assessments should be determined by the wound characteristics observed at the previous dressing change, the significance of wound changes from one assessment to the next, as well as the physician's or other practitioner's orders. Patient interventions should be implemented based on the baseline and subsequent wound assessment data (see Practice Point: When to Reassess a Wound).

Practice Point

When to Reassess a Wound

Assessment provides indicators of successful treatment interventions and attainment of achievable outcomes and guides decisions about product changes. Reassess the patient's wound:

- before and after any surgical or specialized procedures
- weekly or with any change in the patient's activity level for a pressure injury[3]
- if the wound noticeably deteriorates
- if the wound becomes odorous, has new purulent exudate, or becomes more painful
- upon observing any other significant change in the condition of the wound, including at the time of transfer or discharge
- on readmission/returned from another facility.

Although wound assessment needs to be in compliance with the regulatory requirements specific to the care setting, no written standard exists outlining the type and amount of information to include

in a wound assessment. Likewise, no single documentation chart, tool, or electronic medical record (EMR) has been designated as the most effective. Banfield and Shuttleworth found that wound assessments were documented significantly more frequently when an assessment chart or form is used and that using a chart or form improves the nurses' assessment skills.[14] The best assessment form is one that is understood used consistently by the facility's staff. Forms that can be completed easily and quickly are more likely to be used on a regular basis. If the staff finds a form too long or difficult, that form is less likely to be used.

Paper forms, in most healthcare settings, have been replaced with digital technology and the mandated electronic medical records (EMRs). Applying digital technology to everyday practice can be challenging. If the wound assessment section of the EMR has been set up by clinicians who work in wound care, then all parameters of the assessment will be monitored and documented, for the benefit of the patient. EMR technology continues to improve yearly. We have the ability with mobile apps to put evidence-based practice guidelines in the hands of clinicians to be used at the bedside and to support the decision-making process.[15] To further assist providers in tracking numerous wound variables, wound electronic medical record (WEMR) databases have also been developed and modified over the years as tools to collate the key information used during wound assessment and treatment.[16,17]

The digital wound care phenomenon is not confided to just the United States. Other countries are also using the latest software to collect data and complete analysis to improve patient care and establish best practice protocols.

Comprehensive Assessment

A complete, comprehensive initial assessment of the individual with a wound includes complete medical and social history, patient/significant other's goals of care, factors that may affect healing, a vascular assessment with extremity wounds, laboratory assessments as needed, nutritional status, pain, risk for developing additional wounds, psychological health and cognitive status, social and financial support systems, functional capacity (related to repositioning, posture, need for assistive equipment or personnel), pressure redistributing ability, support surface availability, knowledge of prevention and treatment, as well as ability to adhere to the treatment plan.[1,3] A minimal wound assessment should include a thorough assessment of the whole patient, identification of the cause of the wound, and wound characteristics such as type of wound, location, size, depth, exudate and tissue type(s) present, and periwound condition.

Wounds can be classified using several different approaches. The partial- versus full-thickness model is used primarily by physicians and clinicians for wounds other than pressure injuries. Damage to the epidermis and part of the dermis constitutes a partial-thickness wound. Abrasions, skin tears, blisters, and skin-graft donor sites are common examples of partial-thickness wounds. Full-thickness wounds extend through the epidermis and dermis and may extend into the subcutaneous tissue, fascia, and muscle. Partial-thickness wounds heal by regeneration with resurfacing or re-epithelialization. Full-thickness wounds heal by repair (secondary intention) with the formation of granulation tissue, contraction, and, finally, re-epithelialization, which of course requires a longer time period for healing.[5]

Pressure injuries and neuropathic ulcers have their own staging and classification systems to indicate the depth of injury and healing methods. Use the specific system for the type of wound (see Chapter 13, Skin–Challenging Conditions; Chapter 14, Pressure Injuries; and Chapter 18, Diabetic Foot Ulcers).

Sibbald and colleagues have created and validated two different screens to assess the feet of persons without diabetes[18] and those known to have diabetes at risk for developing a diabetic foot ulcer[19,20] (See Tables 6-2 and 6-3). Because some people may be unaware that they have diabetes, the Healthy Foot Screen can assist in early detection of skin changes.[18] When the interprofessional team implemented the GENERIC Simplified 60-Second Screen for the High-Risk Diabetic Foot in persons with diabetes, amputations were reduced by 68%.[19] Based on continuing research by Woodbury et al., in 2015, fissure was removed from the original 2012 screening tool[20] (see Table 6-3). A video that supplements the published articles on how to use this screening tool is available free from the WoundPedia web site (www.woundpedia.com).

Table 6-2 Healthy Foot Screen ©2017

Healthy Foot Screen ©2017

Name: _____

DOB (dd/mm/yy): ____/____/____ **Gender: M □ F □**

ID#: _____ **Phone #:** _____

Completed by: _____ **Date of Exam (dd/mm/yy):** ____/____/____

Check both feet and circle responses where applicable

HISTORY	**1. Is the patient known to have diabetes mellitus type 1 or type 2?** *(If yes, perform simplified 60 second screen)*	Yes		No	
	2. Is the patient currently a smoker? *(If yes, counsel on smoking cessation)*	Yes		No	
	3. Does the patient have neuropathy? Ask about burning, stinging, shooting or stabbing in either foot *(Any person with neuropathy-perform simplified 60 second screen)*	**RIGHT** Yes	No	**LEFT** Yes	No
	4. Has the patient had a previous foot ulcer (U) or amputation (A) on either foot? *(If yes, perform simplified 60 second screen)*	Yes U | A	No	Yes U | A	No
INFECTION	**5. Are there signs of toenail fungal infection/inflammatory changes e.g. trauma, psoriasis?** *(If yes, should be treated)*	**RIGHT** Yes	No	**LEFT** Yes	No
	6. Are toe webs abnormal, especially 4/5th toe web? *(If yes, should be treated)*	Yes	No	Yes	No
	7. Is the bottom of the foot dry? *(If yes, should be diagnosed and treated)*	Yes	No	Yes	No
STRUCTURAL CHANGES ***List specific changes in the comments section below***	**8. Is there a significant bony abnormality or associated change?** *(Any significant bony change should trigger a foot specialist referral)*	**RIGHT** Yes	No	**LEFT** Yes	No
CIRCULATION	**9. Can you feel a foot pulse?** *(Absent dorsalis pedis & posterior tibial pulse = vascular laboratory assessment/ referral)*	Yes	No	Yes	No
	10. Any pitting edema of the feet/ ankle? *If yes, should be diagnosed and treated*	Yes	No	Yes	No

Refer to enabler for more information. Local referral patterns may vary depending on available resources

Other Comments/Abnormalities

_____
_____
_____
_____
_____

Recommendations/Treatment

_____
_____
_____
_____
_____

	ITEMS ON SCREEN	ACTION
1	**DIABETES** **"Yes" if patient is known to have diabetes mellitus type 1 or type 2**	• **Monitor HbA1c** every 3 months • **Perform The simplified 60 second screen**[1] (Accessible at www.WoundPedia.com)
2	**SMOKING** **"Yes" if patient is a current smoker** **Each cigarette:** ↓ skin oxygen 30%/ 1 hr. **Smoking:** ↓ host response = delayed healing [2]	• **Review smoking cessation options**
3	**NEUROPATHY** **"Yes" if burning, stinging, shooting or stabbing pain on either lower extremity** • **Types of neuropathy:** Sensory, Autonomic, Motor • **Common causes: Diabetes** (neuropathy occurs earlier with poor glycemic control), **B12 deficiency, Trauma, Toxins, Chemotherapy, Cancer, Kidney disease, Idiopathic**	• **Perform The simplified 60 second screen** • **Needs accommodative footwear** • **Optimize HbA1c**
4	**PREVIOUS ULCER OR AMPUTATION** **"Yes" if previous ulcer and/or amputation from history is reported or observed** *Indicates a high-risk foot. Callous = pressure Blister = shear and friction*	• **Perform The simplified 60 second screen** • **Check footwear** • **Refer-Foot specialist** (Chiropodist/Podiatrist)
5	**TOENAIL FUNGAL INFECTION (Onychomycosis)/INFLAMMATORY CHANGES**[3] **"Yes" if nails have distal streaks, white/yellow/brown discolouration, subungal debris** • **Onychomycosis can be suspected if any or all of the above are present** **- 50% of nail disorders are fungus** • **Inflammatory changes e.g. due to trauma, psoriasis, lichen planus**	• **Perform nail clipping** (should include subungal debris) & **confirm diagnosis of onychomycosis with:** - A positive microscopic result for at least one toenail along with clinical presentation AND/OR - A positive culture for fungus • **Treat according to % of nail involvement:** > 60% = Oral Terbinafine 20-60% = Topical Efinaconazole +/- Oral Terbinafine (> 3 nails) < 20% = Topical Efinaconazole
6	**ABNORMAL TOE WEB** **"Yes" if toe web (particularly 4-5th web space) is dry, scaly (think fungus) OR wet, macerated (think secondary bacterial infection)**	• **Take scraping (dry, scaly) or bacterial swab (wet, macerated)** • **Treat** with antifungals and/or antimicrobials
7	**DRY FOOT** **"Yes" if dryness in a 'Moccasin' distribution (dryness extends to sole, heel & sides of foot)** • **Diagnose cause** (rule out Neuropathy) – Dryness in moccasin distribution– think fungus – Dryness on plantar surface – think neuropathy	• **Take scraping** • **Treat accordingly**
8	**COMMON STRUCTURAL ABNORMALITY** **"Yes" If deformity and/or abnormality in shape or structure of either foot** • **Hammer toe(s)** metatarsal phalangeal joint up, **Claw toe(s)** metatarsal phalangeal joint up + interphalangeal joint down, **Bunion** enlargement of the base of large toe (Hallux Valgus)	• **Check footwear** • **Refer to foot specialist** (chiropodist/podiatrist)
9	**ARTERIAL DISEASE** **"Yes" if any of the pedal pulses are absent** • Palpate Dorsalis Pedis, if absent check for Posterior Tibialis • **Clinical signs:** cold feet, decreased capillary refill, dependent rubour, intermittent claudication	• **If both dorsalis pedis and posterior tibialis pulses are absent = vascular laboratory assessment and/or referral**
10	**VENOUS DISEASE** **"Yes" if pitting edema of lower legs, feet/ankle is present** • Associated with varicosities, hyperpigmentation, lipodermatosclerosis (hard woody change) • If the hard skin extends to the toes and the skin over the second toe cannot be pinched = + ve Stemmer's sign = Lymphedema	• **Diagnose cause and prescribe compression therapy** if no arterial compromise • **Consider Pentoxifylline (Trental)** • **Avoid long periods of standing or sitting** • **Elevate legs and calf-muscle pump exercises**

OTHER ABNORMALITIES/LESIONS:

- **Psoriasis:** Discrete red plaques + silvery scale = look elsewhere, Sterile yellow (new) to brown pustules especially in females that smoke
- **Dermatitis- Nummular**: Annular lesions/ dorsum; **Contact**: Allergic – dorsum, irritant –plantar
- **Cellulitis:** Bacterial infection – painful red warm dermal swelling
- **Callus:** Hyperkeratosis over pressure points
- **Corn or clavus**: Often small; **Hard corn**: small hyperkeratotic spot – on plantar skin; **Soft Corn**: often in the 4th & 5th web space
- **Gouty Tophus:** Painful lesion often on the large toe associated with a white chalk like discharge
- **Morton's Neuroma**: Inflamed plantar nerve, tender nodule at the base of 3-4th toes
- **Plantar Wart:** Has keratotic surface with punctate bleeding points that pushes away skin surface markings
- **BENIGN: Stucco keratosis (SK)**: aging spot (small seborrheic keratosis= SK); **Melanocytic nevus**: pigmented lesion with normal nevus cells; **Dysplastic nevus:** larger size and more variable colour
- **PRECANCEROUS: Actinic keratosis (AK):** red, rough, scaly spot, common in areas of sun exposure; **Cutaneous horn**: thin hyperkeratotic column with variable base e.g. AK + SK, or skin cancer (SCC, Other)
- **SKIN CANCER: Basal Cell Carcinoma**: translucent papules, ulcers with telangiectasia associated with the base; **Squamous Cell Carcinoma**: irregular keratotic papule that may be painful; **Malignant Melanoma**: asymmetry, irregular border, colour variability- black, blue, red, white, > 6mm and evolving

[1]Woodbury MG et al. Tool for Rapid & Easy Identification of High Risk Diabetic Foot: Validation & Clinical Pilot of the Simplified 60 Second Diabetic Foot Screening Tool. PLoS ONE. 2015;10(6):e0125578. doi:10.1371/journal.pone.0125578, [2]Jensen JA. Cigarette smoking decreases tissue oxygen. Arch Surg. 1991; 126(9): 1131, [3]Gupta AK et al. Canadian Approach with a New Transungual Treatment: Development of a Clinical Pathway. J Cutan Med Surg. 2015 Sep-Oct;19(5):440-9

From Persaud, R., Coutts, P.M., Brandon, A., et al. Validation of the Healthy Foot Screen: A Novel Assessment Tool for Common Clinical Abnormalities. *Advances in Skin & Wound Care* 31(4):154-62, April 2018.

Table 6-3 GENERIC-Simplified 60-Second Screen for the HIGH-RISK DIABETIC FOOT 2016

GENERIC- Simplified 60-Second Screen for the HIGH-RISK DIABETIC FOOT 2016

©Sibbald (2012)

Name: _____

ID#: _____ **Phone #:**_____ **Facility:**_____

DOB (dd/mm/yy):_____/_____/_____

Gender: M □ F □ Years with diabetes:_____

Ethnicity: Black □ Asian □ Caucasian □ Aboriginal □ Other □

Date of Exam (dd/mm/yy):_____/_____/_____

CHECK BOTH FEET
(Circle correct response)

YES on either foot is = HIGH RISK

		LEFT		RIGHT	
History	**1.** Previous Ulcer	NO	YES	NO	YES
	2. Previous amputation	NO	YES	NO	YES
Physical exam	**3.** Deformity	NO	YES	NO	YES
	4. Ingrown toenail (thickened nail fold)	NO	YES	NO	YES
Foot lesions *Remember to check 4th and 5th web spaces and nails for fungal infection Remember to check for inappropriate footwear*	**5.** Absent pedal pulses (Dorsalis Pedis and/ or Posterior Tibial)	NO	YES	NO	YES
	6. Active ulcer	NO	YES	NO	YES
	7. Blisters	NO	YES	NO	YES
	8. Calluses (thick plantar skin)	NO	YES	NO	YES
Neuropathy *MORE THAN 4/10 SITES LACKING FEELING = **"YES"***	**9.** Monofilament exam **(record negative reaction):** **a)** Right_____/10 negatives (≥ 4 negatives = Yes)	NO	YES	NO	YES
	b) Left_____/10 negatives (≥ 4 negatives = Yes)	NO	YES	NO	YES
		Total # of YES:____		**Total # of YES:** ____	

PLAN

a) POSITIVE SCREEN- Results when there are one or more "Yes" responses. **Refer to a foot specialist or team for prevention, treatment and follow up**. (Bony deformity, current ulcer, absent pulse are most urgent). These individuals are at increased risk of a foot ulcer and/or infection. Patients should be educated on what changes to observe and report, while waiting for the specialist appointment.

Referral to: _____ **Appointment time:**_____

b) NEGATIVE SCREEN- Results when there are all "No" responses. **No referral required.**
Educate patient to report any new changes to their healthcare provider and re-examine in 1 year.

One Year Date for Re-Examination (dd/mm/yy):_____/_____/_____

Completed By: _____ **Date:** _____

Additional Note:
For **POSITIVE SCREENING**, in addition to referral plan above, **positive risk factors** can be linked to the care recommendations in "Root Risk Classification and Follow- Up Guide" table on the bottom of reverse side. Local referral patterns may vary depending on expertise and available resources.

GUIDELINES FOR USE:
GENERIC-Simplified 60-Second Screen for the HIGH-RISK DIABETIC FOOT 2016

General Instructions:
This diabetic foot screening tool is designed to identify individuals with high-risk diabetic feet. The screening tool is a simplified 60-second assessment for each foot, to be implemented by any healthcare provider. Preparation involves having a 5.07g monofilament available and asking patient to remove their shoes and socks.

Normal screening findings are indicated as "No" (not requiring referral) and abnormal screening findings are indicated as "Yes" (requiring referral). Generation of a list of local reputable foot specialists and/or teams for referring is recommended.

Screening involves:

- Inform patient about the simplified 60-second screening and explain the reason for the examination.
- Fill in patient's demographic data in top left section of screening tool.
- Assess both feet. Circle either a "Yes" or "No" response to questions 1-9.
- **Any "Yes" response requires follow up or a referral to a foot specialist and/or team.**

Question	"Yes" Response
1	**"Yes", if previous ulcer from history is observed:** Ask the patient and assess both lower legs and feet for the presence of a healed ulcer as evidenced by scar tissue.
2	**"Yes", if previous amputation:** of digit(s), foot or limb is observed.
3	**"Yes", if deformity:** and/or abnormalities in shape or structure of either feet is observed (bony prominences/ hammer toes).
4	**"Yes", if ingrown toenail present.** Inspect distal corners for embedded nail and/or thickened nail fold skin.
5	**"Yes" If absent pedal pulses** (palpate Dorsalis Pedis and if absent check Posterior Tibial). A yes answer requires absence of both pulses.
6	**"Yes", if active ulcer(s) present:** Openings in the skin with a dermal or deeper base.
7	**"Yes", if blisters present:** Observe for fluid (serum, blood or pus) under intact skin surface.
8	**"Yes", if callus present (thick scale on plantar skin):** Assess and inspect for presence of thick areas of keratin on the bottom or sides of feet and toes.
9	**"Yes" of monofilament Exam identified 4 or more negative reactions (lack of feeling)**: Follow the monofilament exam instructions below. Each foot is examined separately.

Steps for Monofilament Test for Neuropathy:
- Show and touch monofilament to patient's arm or upper leg.
- Ask the patient to close their eyes and say yes when they feel the monofilament.
- Touch monofilament until filament bends in a letter "c" shape, assessing all 10 areas on diagram (Do not test over calluses, scars or ulcers)

- Lack of feeling (4 or more out of 10) - indicates a negative reaction = Neuropathy = "YES" on screening tool

Foot Risk Classification and Follow-up Guide

Assessment Findings ↓	Risk	Follow Up (mths)	Prof. Nail Care	Shoes	Orthotics + Diabetic Socks	Activity
No Neuropathy	0	12	-	Well fitting	Well fitting shoes	As able
Neuropathy	1	6	+/-	Professional fit	Custom full contact	As able, monitor, guided by foot exam
Deformity	2a	3-4	+/-	+/- custom fit	Custom full contact	Avoid excessive walking, √ non-impact exercises
Peripheral Vascular Disease	2b	3-4	+	Professional fit	Soft full contact	Dependent on ischemic pain, √ non-impact exercises
Ulcer Hx or Active ulcer	3a	1-2	+/-	Professional fit	Custom fitted	Activity dependant on exam, √ non-impact exercises
Hx Amputation	3b	1-2	+/-	Special clinic (assessment) Modified footwear	Specialized clinic: amputation/prostheses, +/- walking aid	Based on tissue tolerance, √ non-impact exercises

Modified from International Diabetes Federation, International Working Group on the Diabetic Foot 2007

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Assessing the severity of a burn is a two-part process and differs from an open wound. Burn injuries are described by the extent of the body burned using one of several methods for estimating burn size, such as the rule of nines[21] or the Lund and Browder Chart[22] (Fig. 6-2). The depth of a burn injury is described by clinical observation or via surgical excision of the anatomic layer of the skin involved (e.g., superficial, partial-thickness, full-thickness, or subdermal burns).

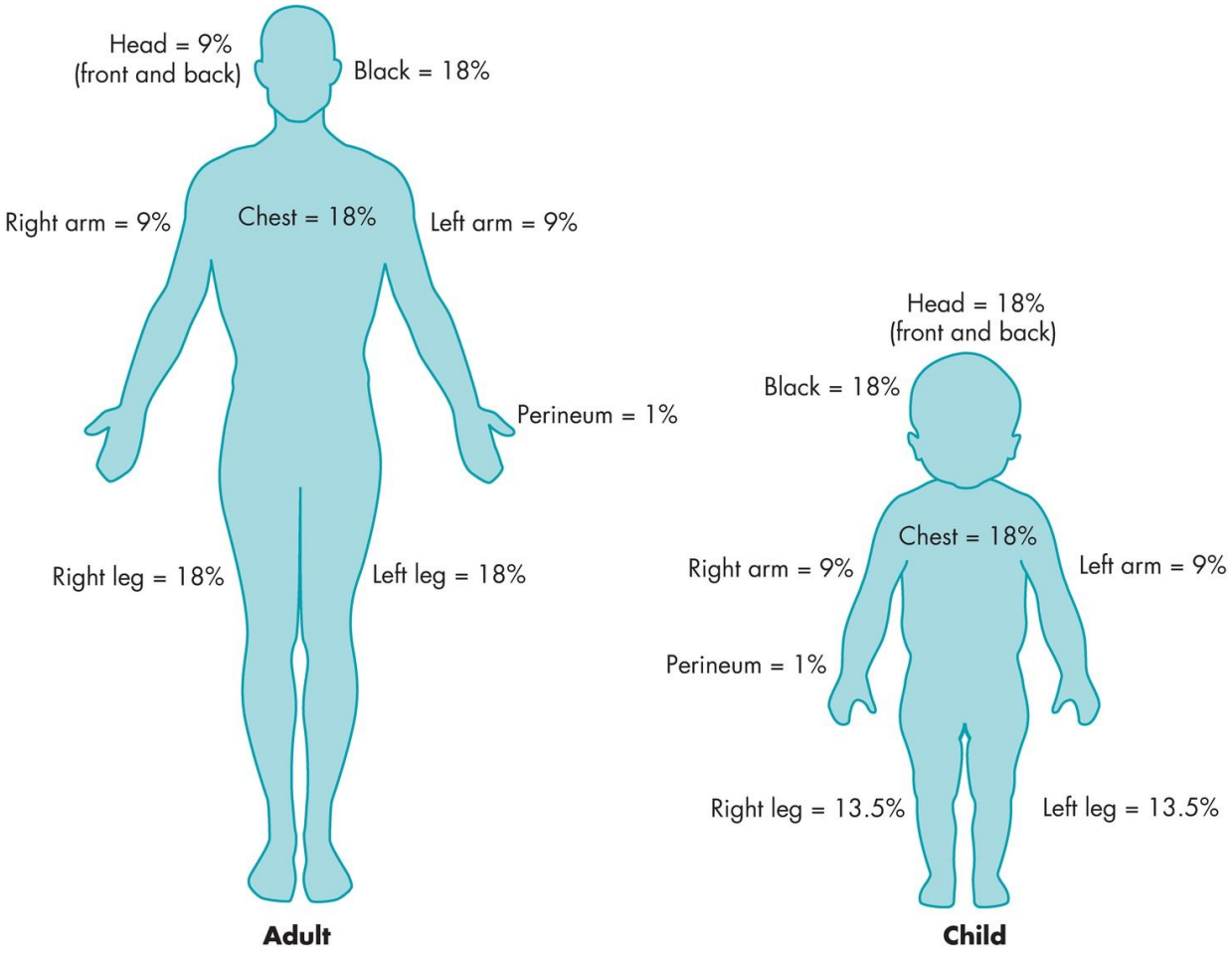


Figure 6-2. The rule of nines. The rule of nines estimates the amount of body surface that has been burned. In adults, the body is divided into sections of 9% or multiples of 9%. The percentages used in the rule of nines differ between adults and children.

Obviously, there are many parameters to consider when performing a comprehensive wound assessment. Each clinical agency needs to develop a protocol that all clinicians should learn and follow to ensure consistency of assessment and documentation whether using a paper chart or an EMR. Whether using stage/category or partial- and full-thickness terminology, the one constant is clinical assessment. Assessment data give the healthcare provider a mechanism by which to communicate, improve continuity among disciplines, and establish and modify appropriate treatment modalities.

Elements of a Wound Assessment

In 1992, Ayello developed a mnemonic for pressure ulcer assessment and documentation.[23] The mnemonic has been adapted (Box 6-2) for use with any type of pressure injury or wound to provide a thorough look at the parameters that complete and enhance an assessment. It provides a support structure for clinical decision-making regarding ongoing assessment and reassessment and may be used in any practice setting according to the guidelines set up by your facility. This assessment chart may be used daily, weekly, or monthly. It's simple, is fast and can be further adapted to fit individual use. It can also be utilized as a valuable tool for setting up the categories needed in the wound electronic medical record. It is always wise to have a hard/paper copy for times when the power is lost during bad weather or when the computer system fails. Old practices can still be used to safeguard care documentation.

Box 6-2 Pressure Injury and Wound Assessments Chart

PATIENT'S NAME: _____

AGE: _____ ☐ M ☐ F

ASSESSMENT DATE:_____ TIME:_____

A Ascertain Wound Etiology: Identify etiology and number of each type of wound in the space provided

☐ Surgical _____
☐ Arterial _____ ☐ Venous _____
☐ Pressure Injury(s) _____
☐ Neurotrophic/DM ulcer _____
☐ Other _____
☐ Skin Tear(s) _____ ☐ Type 1 ☐ Type 2
☐ Type 3
☐ Moisture-Associated Skin Damage _____

Anatomic location of wound

☐ Upper/lower chest ☐ Abdomen ☐ Incisional
☐ Face ☐ Head ☐ Back ☐ Ear ☐ R ☐ L
☐ Elbow ☐ R ☐ L ☐ Arm ☐ R ☐ L
☐ Sacrum/coccyx ☐ Ischium ☐ R ☐ L
☐ Gluteal Fold
☐ Leg ☐ R ☐ L ☐ Trochanter ☐ R ☐ L
☐ Foot ☐ R ☐ L ☐ Foot ☐ R ☐ L
☐ Toe(s) Specify _____
☐ Lateral malleolus ☐ R ☐ L
☐ Medial malleolus ☐ R ☐ L
☐ Heel ☐ R ☐ L
☐ Other _____

Age of wound _____

☐ Acute date of onset _____
☐ Chronic date of onset _____
Date of onset : _____
_____ days or _____ months patient has had the pressure injury or wound

S Size

_____ cm length _____ cm width _____ cm depth

Shape

☐ Oval ☐ Round ☐ Linear ☐ Irregular
☐ Other _____

Stage/Grade/Category/Classification

☐ **Pressure Injury (PI)**
☐ 1 ☐ 2 ☐ 3 ☐ 4 ☐ DTI
☐ Unstageable—necrotic tissue

☐ **Medical Device–Related Pressure Injury**
☐ 1 ☐ 2 ☐ 3 ☐ 4 ☐ DTI ☐ Unstageable

☐ **Mucosal Membrane Pressure Injury** (cannot not stage using NPIAP system)

☐ **Wagner Ulcer Classification—Diabetic Foot Ulcer**
☐ 0 ☐ 1 ☐ 2 ☐ 3 ☐ 4 ☐ 5

S sinus tract, tunneling, undermining, fistulas

Sinus tract ☐ none ☐ present at _____ o'clock _____ cm
Tunneling ☐ none ☐ present at (narrow tracts under the skin) at _____ o'clock _____ cm
Undermining (bigger area [than tunneling] of tissue destruction—area is more like a cave than a tract) ☐ none ☐ present at _____0'clock
Fistulas ☐ none ☐ present at _____

E Exudate

Amount ☐ None ☐ Scant ☐ Moderate ☐ Large
Color ☐ Serous ☐ Serosanguineous ☐ Sanguineous ☐ Green ☐ Brown ☐ Other _____
Consistency ☐ Clear ☐ Purulent
Odor ☐ Present ☐ None

S Sepsis

☐ Local ☐ Systemic ☐ Both ☐ None

S Surrounding skin

☐ Intact ☐ Swollen ☐ Induration ☐ Discolored ☐ Dark ☐ Temperature ☐ Warm ☐ Cool ☐ Dry ☐ Moist ☐ Other _____

M Margins

☐ Attached (edges are connected to the sides of the wound)

☐ Not attached (edges aren't connected to the sides of the wound)

☐ Rolled (edges appear rounded or rolled over [epibole])

Maceration

☐ Present _____ cm,
location _____

☐ Not present

E Erythema

☐ Present ☐ Not present

Epithelialization

☐ Present ☐ Not present

Eschar (necrotic tissue)

☐ Yellow slough ☐ Black ☐ Soft ☐ Hard
☐ Stringy ☐ Other _____
Area around eschar is: _____
☐ Dry ☐ Moist ☐ Reddened

N Necrotic tissue

☐ Present ☐ Not present
☐ Yellow slough ___% ☐ Black ____%
Percentage of wound (check closest percentage):
☐ 100% of wound ☐ <75% ☐ >75%
☐ <50% ☐ >50% ☐ <25% ☐ >25%
☐ Other _____%
☐ Soft ☐ Hard ☐ Stringy

Nose

☐ Odor present in wound ☐ Odor not present

T Tissue Bed

Granulation tissue: ☐ present ☐ not present
Epithelialization: ☐ present: _____ cm
☐ not present
☐ Surgical incision approximated
☐ Sutures/staples intact

Tenderness to touch (pain)
☐ No pain ☐ 0
☐ Pain present: (0 being no pain, 10 being intense pain)
☐ 1 ☐ 2 ☐ 3 ☐ 4 ☐ 5 ☐ 6 ☐ 7 ☐ 8 ☐ 9 ☐ 10
Pain present:
☐ On touch ☐ All the time
☐ when patient moving
☐ Only when performing wound care
☐ Only when irrigating/cleansing the wound
☐ Only when changing the dressing
☐ Other _____
Patient getting pain medication: ☐ Yes ☐ No
Pain management effective ☐ Yes ☐ No

Tension
☐ Tautness, hardness present ☐ Not present

Temperature
☐ Skin warm to touch ☐ Skin cool to touch
☐ Normal

S *Status of Wound*:
☐ Surgical incision open ☐ Surgical incision approximated ☐ Sutures intact ☐ Staples intact
☐ Improved: date _____ ☐ Unchanged: date _____
☐ Healing: date _____ ☐ Unchanged
☐ Deteriorating: * date _____
* Notify Physician
☐ Supportive therapy
☐ compression ☐ off loading _____
☐ Pressure redistribution device(s)
☐ other
☐ Patient's perception on quality of life _____
☐ Case management/social services needs
☐ Nutrition consultation requested
☐ PT ☐ OT
☐ Referral to other departments _____

Initial assessment:
Signature _____ Title _____ Date _____
Reassessment:
Signature _____ Title _____ Date _____

*Notify Physician. If you check the box for deteriorating the asterisk refers to then you must notify the physician.© 2019 Revised Ayello and Baranoski.

Location and Age of Wound

Wound location should be documented using the correct anatomical terms—for example, right trochanter rather than right hip. Include an anatomical figure or diagram of the human body, with the wound's location noted in your assessment record to provide complete admission documentation. Documenting wounds on the buttock presents challenges for clinicians. Where does the gluteal region begin and end? Locations are often not documented or documented incorrectly (see Fig. 6-3). This schematic created by Berke/Morris©, a wound ostomy and continence nurse, teaches the correct terminology for the buttock area. It is a useful tool to enhance documentation skills and clarify location of pressure injuries, moisture-associated skin conditions, or other wound injuries.

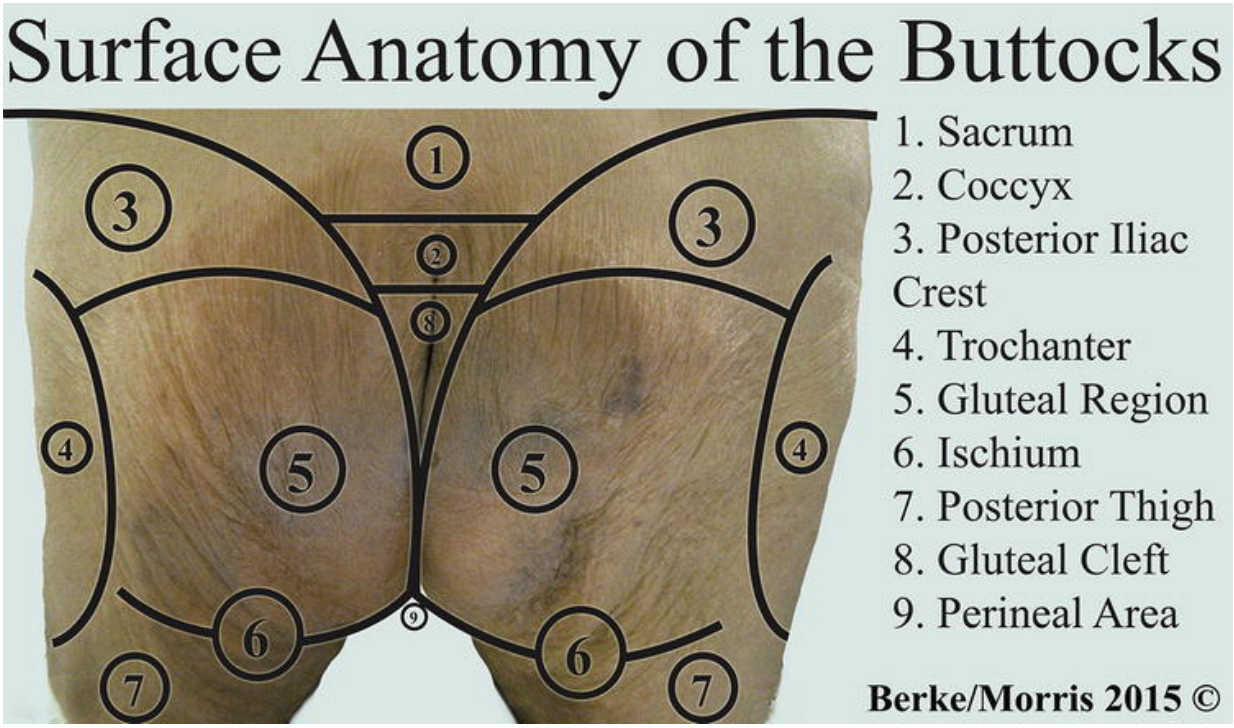


Figure 6-3. Surface anatomy of the buttock. Used with permissions from Berke/Morris 2015©.

If there are two or more wounds near one another, they should be labeled and numbered for clarity (e.g., 1a, 1b, upper, lower, etc.). It is particularly important to note how long the patient has had the wound. CMS used to require that the date of the oldest stage 2 pressure injury be recorded on Minimum Data Set 3.0 (M0300B.3)[24] in the long-term care setting. As of October 2018, this is no longer required in LTC.[24]

Are you dealing with a new, acute wound or a wound that has failed to heal for several weeks or months? Time is important but isn't the sole determinant of acute versus chronic wound status. Although 30 days is often used for designation as chronic status, the more important criterion is whether or not the wound is making progress toward healing.[5,24]

In addition to wound duration, documentation of the etiology of the wound, if known, is important. For example, if a patient reported that she spilled hot coffee on her amputated stump, causing a blister that evolved into a full-thickness wound due to trauma and insufficient arterial supply, it would be incorrect to classify the wound as a pressure injury. Remember, it is important to know what caused the blister. It is not unusual for an individual with diabetes mellitus to have a neuropathic/vascular ulcer or to have multiple ulcers of more than one origin.

Wound Size and Stage/Category

The joint National Pressure Injury Advisory Panel (NPIAP)–European Pressure Injury Advisory Panel (EPIAP) classification system[3] is only intended for use in staging/categorizing pressure injuries. It was revised in 2009 to include four numerical stages with two additional categories for use in the United States that incorporate suspected deep tissue injury and unstageable injuries into their own separate categories.[3] The staging/categorization system addresses the depth

of tissue damage in numerical stages/categories 1 through 4, unstageable, and deep tissue injury. Any pressure injury covered with eschar or necrotic tissue is unstageable, including in long-term care where CMS now requires that it be documented on MDS 3.0 under the unstageable section M0300F in the United States.[13,25] Reverse staging is no longer required in the long-term care setting[13,25] and is never recommended.[26] In 2016, NPIAP revised the staging system once again and added two new definitions for etiologies (types) of pressure injuries, medical device–related pressure injury (MDRPI) and mucous membrane pressure injury (MMPI).[27] See The Pressure Injuries in Chapter 14 for more details.

Partial-thickness wounds heal fairly quickly (days to weeks) as they involve the epidermis and extend into, but not through, the dermis. Full-thickness wounds penetrate through the fat and involve the muscle, tendon, or bone and take longer to heal (Fig. 6-4A and B). Use the correct classification system for the specific wound type, for example, Meggitt-Wagner for neuropathic (see Chapter 18, Diabetic Foot Ulcers) or International Skin Tear Advisory Panel (ISTAP) for skin tears (see Chapter 4, Skin: An Essential Organ).

WOUND HEALING
A

Figure 6-4. (A) Necrotic, unstageable pressure injury. Shown here is a pressure injury that's unstageable because its base is covered with eschar. Be sure to measure the pressure injury's length, width, and percent and type of necrotic tissue. Document your findings. **(B)** Necrotic calcaneal wound with exposed tendon. Wound is 50% covered in necrotic tissue with a small amount of granulation tissue. Tendon is readily exposed. Courtesy of Jeff Niezgoda, used with permission.

Practice Point

Even though an ulcer is necrotic and unstageable, you still need to document the wound size for length, width, percent and type of tissue present, exudate, and odor.

Wound Measurement

Measurement of a wound is an important component of wound assessment and provides valuable information on wound progression or nonprogression as well as assessment of the effectiveness of clinical interventions. Wound measurement is particularly important in determining clinical effectiveness for research purposes. Consistency and accuracy in how the wound is measured are important for meaningful comparisons to determine changes in the wound over time and for comparing the effectiveness of various treatments. Consistency is best assured when the agency develops and disseminates a protocol for wound measurement that staff can follow. It is also important to use consistent patient positioning every time a wound is measured.[3]

Wound measurement methods can be simple or sophisticated or two-dimensional (wound surface area) or three-dimensional (wound volume). A variety of systems are used to measure wounds and assess healing. These include wound tracing, width and length measurements, computerized wound-documenting systems (which can be one-, two-, or three-dimensional), and digital photography.[1,28,29] Changes in wound measurements, such as a decrease in size, are used as an indicator of healing. Surgical incisions can be measured using length (e.g., “incision line is 8 cm long”). Wounds should not be measured using objects, such as a dime or half-dollar, but rather should be measured in centimeters or millimeters depending on the size of the wound.

Area

The simplest and most common method of wound measurement is the linear method using a paper or plastic ruler marked in centimeters and millimeters. The NPIAP Position on Wound Area Measurement, outlined in a 2008 study by Langemo and colleagues,[29] is to measure the greatest head-to-toe length and the greatest side-to-side width perpendicular (90-degree angle) to each other.[2,29] If this method is used consistently, then measurements over time should become more reliable and comparable (Fig. 6-5). Linear measurement is inexpensive, is readily available, causes little to no discomfort, and is used frequently by most clinicians.[3,29] However, use caution with this method, as it assumes that the wound area is a rectangle or square, which is rarely ever the case, and nearly always overestimates the size of the wound.[27] Regardless of which method is used, what's most important is to have an agency protocol that the staff understands and that is being implemented correctly and consistently.

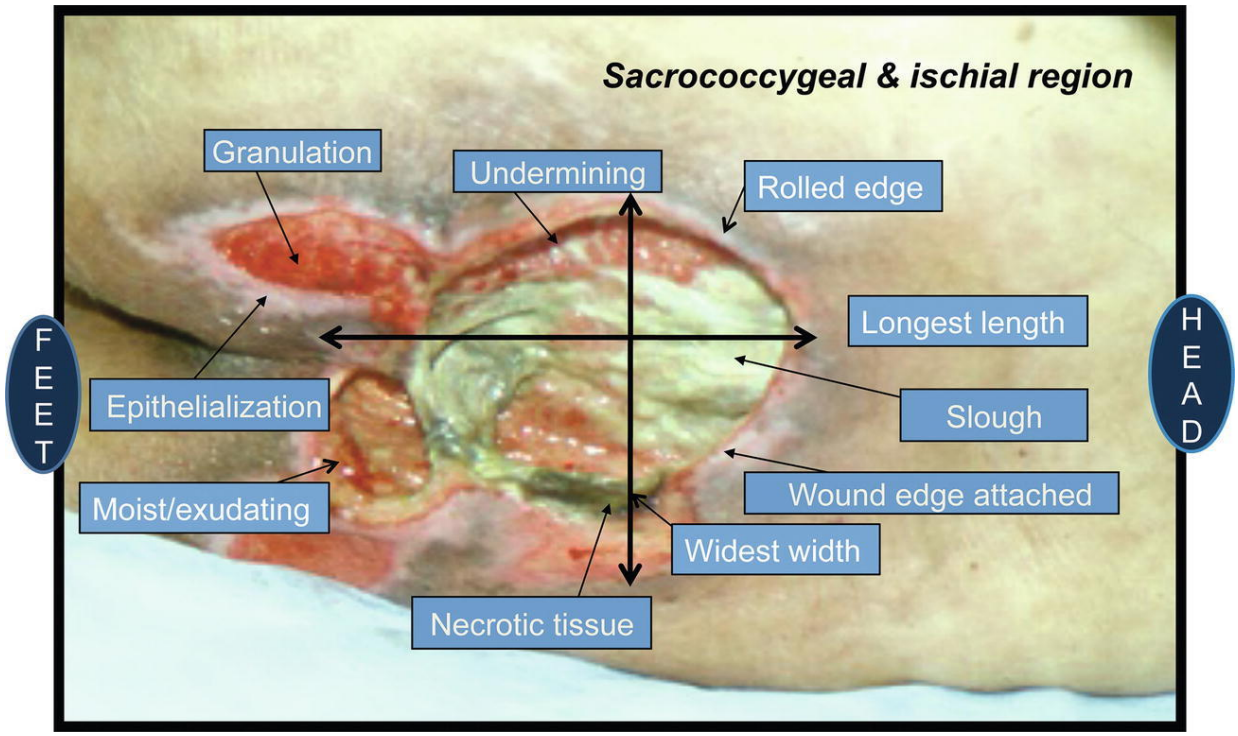
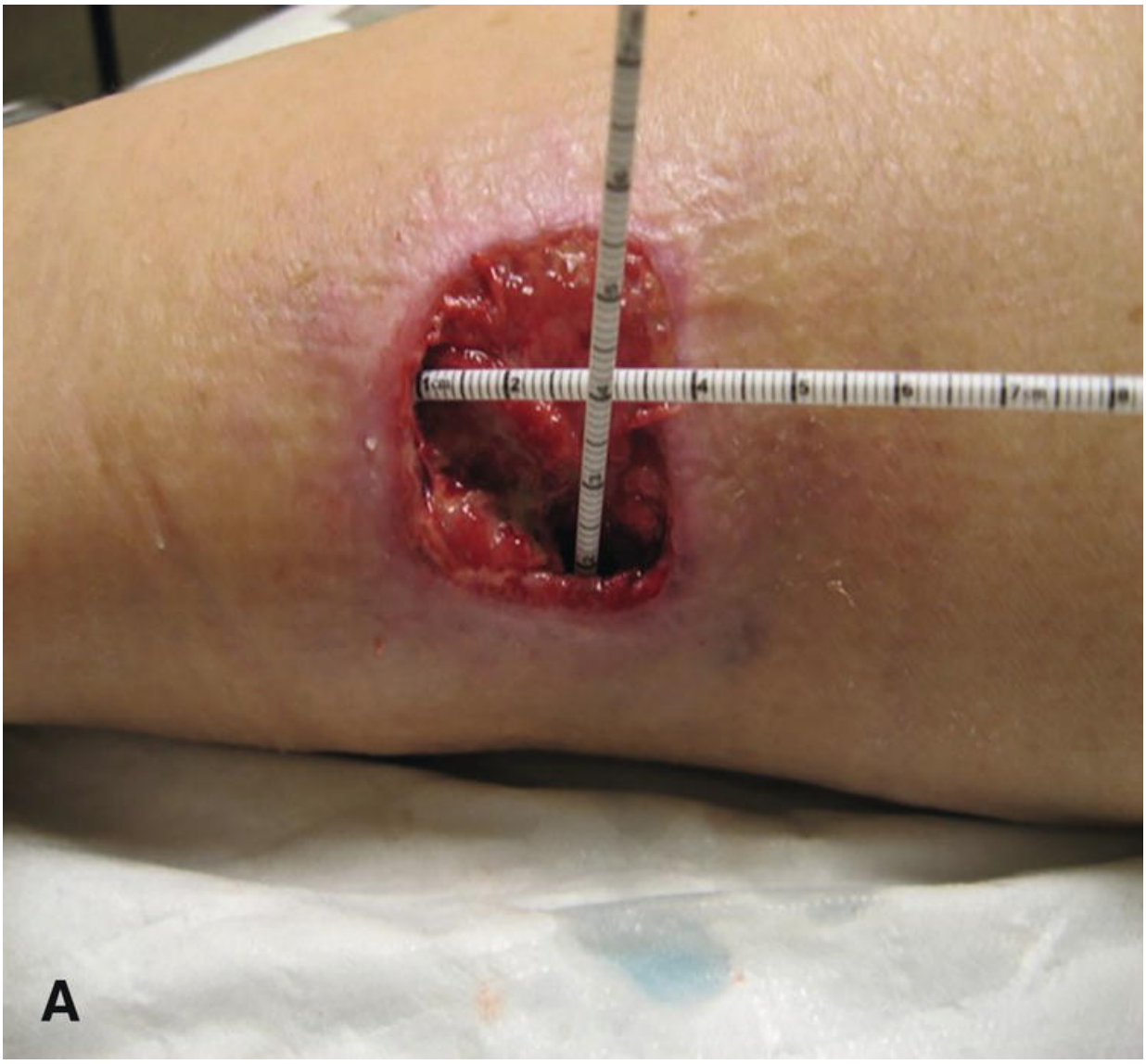


Figure 6-5. Wound terminology. Using current terminology is imperative for accurate assessment.

This photograph labels the wound's characteristics as well as its length and width.

Another way to measure area is to multiply length by width in square centimeters (cm^2). This adds a third dimension of depth, which is then added to the linear measurement if desired.[30,31] If the wound is open, the depth can be assessed by placing either a clean cotton-tipped applicator or a centimeter-measuring device into the deepest part of the wound, marking it, and then measuring it upon removal (Fig. 6-6A and B).


A

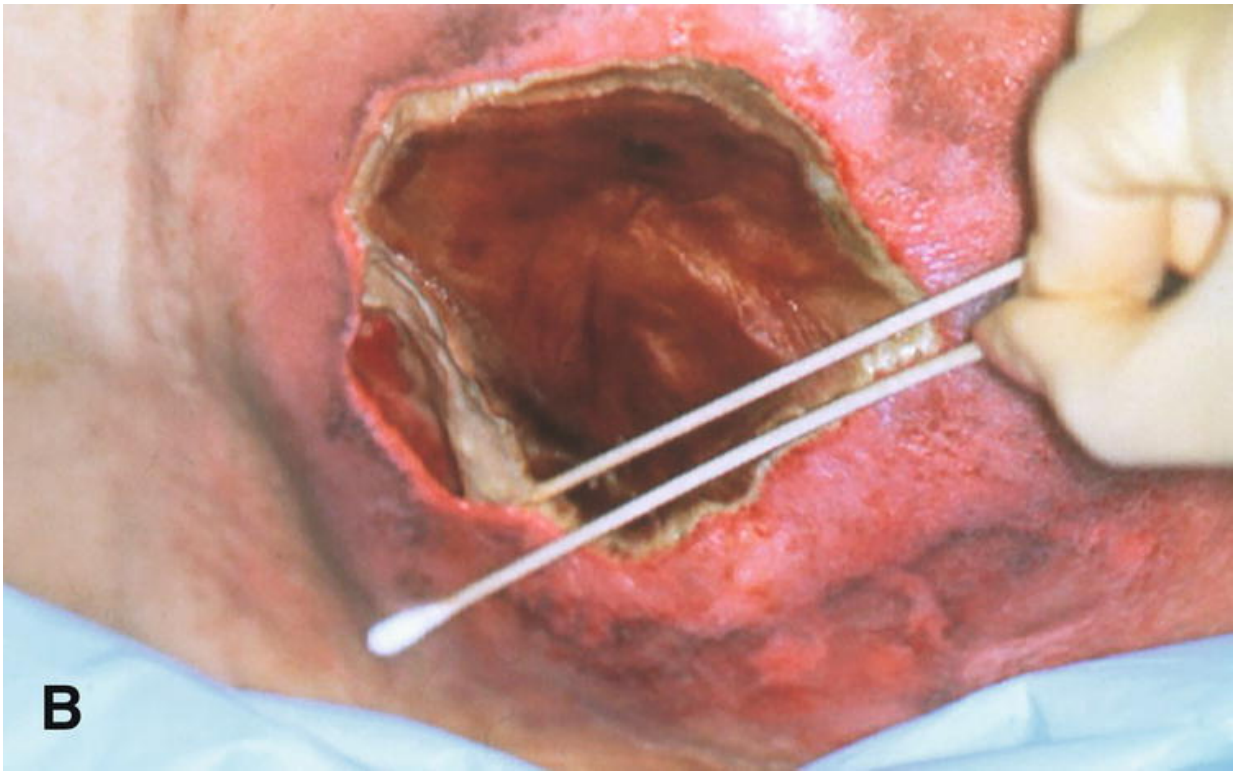


Figure 6-6. (A) Determining wound depth. The top figure is measured using prelabeled plastic applicators frequently used in outpatient settings. (Courtesy of L. Montoya.) **(B)** The depth of wound can be measured by placing a sterile, premoistened (with normal saline or sterile water) cotton-tipped applicator into the wound and comparing the marked area against a centimeter-measuring device (Courtesy of S. Baranoski.).

Planimetry is a method where a wound tracing is made on metric graph paper with a 4- or 8-cm grid. The completed squares, within the traced wound edges, are then counted to yield an approximate area in square centimeters.[32] Minimal training is needed to use this method, the acetate tracing medium is inexpensive and disposable, and the wound area can be determined immediately.[30]

The wound area can also be measured noninvasively by using various types of digital imaging. Stereophotogrammetry (SPG) measures the area using a digital camera and computer software. A target plate is placed within the plane of the wound to be photographed. The digital photo is then downloaded to the computer screen where the wound edges are traced, along with the length and width, using a computer-pointing device or mouse. The software automatically calculates the area as well as the length and width.[30] A color picture of the wound along with the measurements taken during each visit can be printed on a chart sheet for the patient record. The WoundVision Scout® is another method that has been documented as valid and reliable. The Scout is a handheld device that is noncontact and nonthermal and measures L and W and allows for outlining of perimeter for accurate area measurement.[32] These methods allow for accurate, reproducible measurements of irregular wounds and are noninvasive.[30–33] Another technology uses structured lighting for 3D measurements of wound area, depth, and volume. Trisecting lasers are used as a range finder and to provide planimetry to measure wound area.[34]

Volume

As most wounds extend below the skin surface, they are three-dimensional, generally irregular, and, at times, cone shaped. To that end, volume becomes an important variable and needs to be calculated. The most commonly used technique to assess wound volume is to measure the three dimensions of length, width, and depth and multiply those measurements by one another (L × W × D = volume cm^3).[31] Caution should be used, however, as this equation assumes that the base and surface area are the same size, which is generally not the case. The net effect is overestimation of wound volume.

Other techniques include molds, fluid instillations, the Kundin device, and SPG. Molds and fluid installations are imprecise and time-consuming, are uncomfortable for the patient, and can potentially contaminate the wound.[35] The Kundin device is a plastic-coated, disposable, three-dimensional gauge with three arms for measuring

length, width, and depth.[32,36] Wound volume is calculated via a mathematical formula that assumes the shape of the wound lies somewhere between a cylinder and a sphere.[36] Measuring volume using the Kundin device is a convenient, relatively inexpensive, user-friendly technique but includes wound contact.[36] As mentioned previously, SPG measures the depth and area of a wound and inputs that information into software that calculates wound volume using the Kundin device formula. In one study, SPG was found to have the greatest reliability and least error of measurement.[31] When the Kundin device and SPG were compared using wound models, SPG was the more accurate method. However, more research is needed. The key is to select and implement a consistent method to measure the depth.[3] As mentioned, structured lighting using trisecting lasers for 3D measurements to provide planimetry can measure the wound area as well as the depth to provide volume calculations. This newer technology reports excellent interrater and intrarater reliability and has found significant adoption in both clinical and research settings.[34]

Sinus Tracts, Undermining, and Fistulas

Sinus tracts/tunnels, undermining, and fistula formation delay the healing cascade. Intervening early with the appropriate medical and/or surgical and/or nursing actions is paramount to healing these complicated wounds.

Sinus Tracts

A sinus tract (or tunnel) is a course or path of tissue destruction, sometimes called a “tunnel,” occurring in any direction from the surface or edge of a wound. Shear can be a culprit in tunneling. It results in dead space with a potential for abscess formation. A sinus can be distinguished from undermining in that it involves only a small portion of the wound edge; undermining involves a significant portion of the wound edge.[3] The sinus or tunnel has a potential for abscess formation, further complicating the healing process. Sinus tracts are common in dehisced surgical wounds and may also be present in neuropathic wounds, arterial wounds, and pressure injuries. Documenting sinus tracts is an important element in assessment

because it enables the clinician to evaluate potential treatment interventions and to identify reasons for nonhealing. Treatment interventions involve loosely packing the dead space with an appropriate dressing to stimulate granulation tissue production and the contraction process. Document what goes into the tract to see that it is removed during dressing changes. The goal is to close the sinus tract first while allowing the outside of the wound to remain open and fully heal.

Measurement of a sinus tract can be made by inserting a sterile, premoistened, cotton-tipped applicator, a centimeter-measuring device, or a gloved finger into the bottom or end of the tract, marking it, and then measuring it upon removal. This must be done very carefully to avoid injury during measurement (Fig. 6-6B).

Undermining

Undermining is tissue destruction that occurs around the wound perimeter underlying an intact skin; in these wounds, the edges have pulled away from the wound's base (Fig. 6-7). Pressure injuries that have been subjected to a shearing force often present with undermining in the area of the greatest shear. Undermining is also seen when the opening of the wound is smaller than the affected tissue below the dermis and in desiccated wound beds.

A

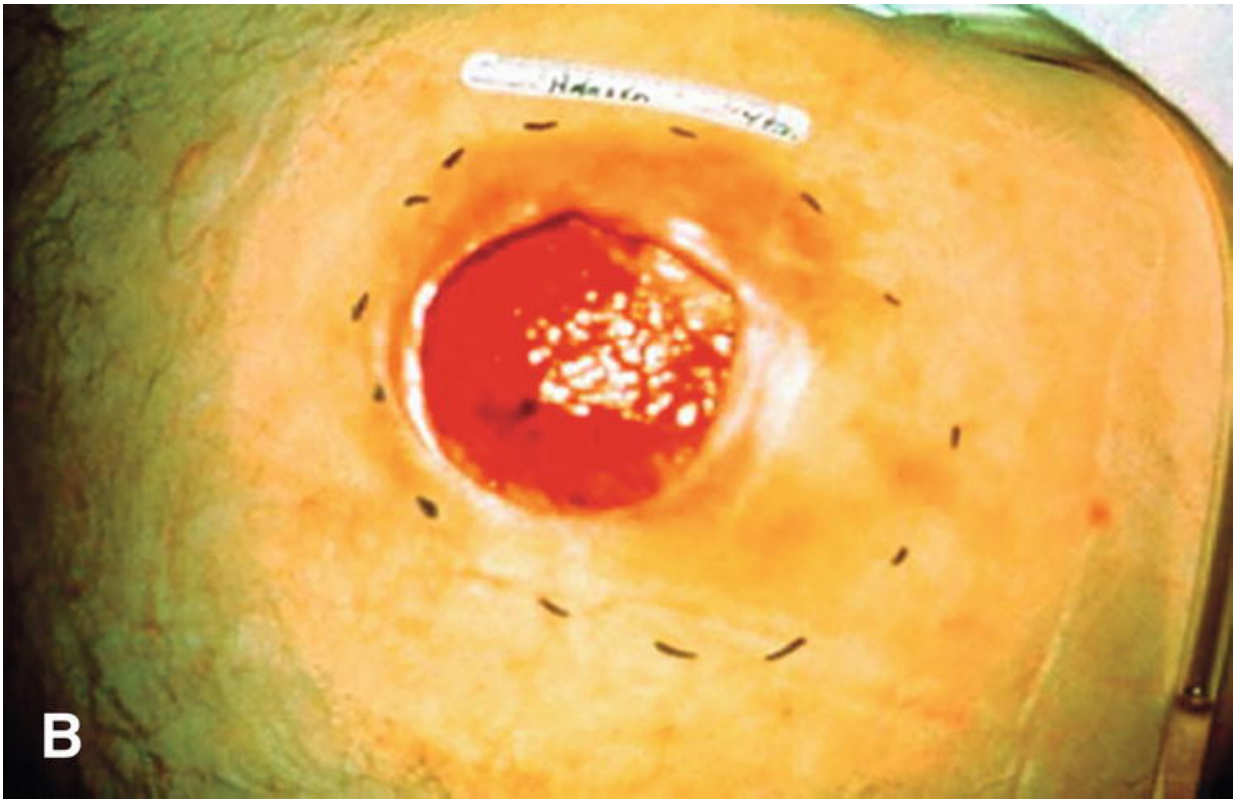


Figure 6-7. (A and B) Undermining, as shown, is tissue destruction that occurs around the wound perimeter underlying an intact skin.

Documentation of the location and amount of undermining is important. Clinicians can document using the clock figure, with the head as the 12-o'clock position (e.g., "undermining from 2 to 6 o'clock measures 3 cm") or using percentages (e.g., "75% of the wound has undermining measuring 2 cm from 12 to 9 o'clock"). Undermining may also be more extensive in one part of a wound than another. This, too, should be documented appropriately. Interventions include loosely packing or tucking all undermined areas to prevent buildup of debris and dead tissue and applying an appropriate dressing, such as hydrogel, gauze, or alginate dressing.

The degree of undermining can be measured using the same method as for sinus tracts.

Fistulas

Fistulas can develop in surgical wounds and in deep, severe pressure injuries. A fistula connects viscous organs together (e.g., a rectovaginal fistula) or connects a viscous organ to the skin (e.g., an enterocutaneous fistula).[37] Fistulas are named by using the point of origin, such as the rectum, and the point of exit, such as the vagina. Management of a patient with a fistula is complex and intense and demands critical thinking and technical skills.[37] Fistulas can take weeks or months to heal. The presence of feces in the area of a fistula is a significant concern. In addition, the patient with a fistula is often malnourished and may require weeks of intense nutritional therapy to improve his or her condition (see Chapter 19 for more on fistulas).

Exudate and Odor

Exudates are accumulated fluids in a wound; these fluids may contain serum, cellular debris, bacteria, and leukocytes. Exudate may appear as dry, dehydrated, dead, or nonviable tissue (nondraining) or be moist and draining. Exudate assessment includes noting the amount (small, moderate, large), color, consistency, and odor.[38] Certain microorganisms, such as *Pseudomonas aeruginosa*, have a characteristic odor. Exudate may indicate that the cause of the wound has not been treated; see Box 6-3 and Table 6-4.

Table 6-4 LOWE© Skin Barriers for Wound Margins: 20-Second Enablers for Practice

Exudate may indicate that the cause of the wound has not been treated (e.g., edema due to venous insufficiency), congestive heart failure is present (look for bilateral involvement and extension above the knee), there is low albumin (malnutrition, kidney or liver disease), or infection is present (check for symptoms or signs).

Periwound skin needs protection from exudate by using absorbent dressings over the wound and protecting the periwound skin. You can choose from four ways to protect the external skin of a wound. Try using this memory jogger to remember them: LOWE© (from Old English, meaning to approve of, prompt, or to humble oneself).

Type	Advantages	Disadvantages
Liquid film Forming acrylate • No sting • Skin preparation, etc.	• Transparent surface that resists removal • Low incidence of reactions	• Some skin sealants may evaporate and dry out • Lack of availability on some institutional formularies
Ointments • Petrolatum • Zinc oxide	• Relatively cheap and easy to apply	• Petrolatum liquefies with heat • Zinc oxide ointment does not allow visualization of underlying wound margin • Ointment vehicle may interfere with the action of ionized silver • Reactions to the adhesive can occur
Windowed dressing Framing of wound margin with protective adhesive: • Hydrocolloid • Film • Acrylate • Silicone, etc.	• Provides a good seal around the wound edge • Some products facilitate visibility of the wound margins	• If seal is compromised, moisture may accumulate under the dressing
External collection devices	• External pouching may help in locations where an external seal is difficult (e.g., perirectal area)	• Devices need to be monitored for external seal *Note: These devices do not replace a search for the cause of the excessive exudate and the need to correct the cause*

© 2006, Ayello and Sibbald. All rights reserved.
Ayello, E.A., Sibbald, R.G. "LOWE© Skin Barriers for Wound Margins: 20 Second Enablers for Practice." *Advances in Skin & Wound Care* 19(5):237, 2006.

Box 6-3 Classifying Exudates

Wound exudate can be classified in two ways—by type or amount.

Type (Color and Consistency)

Exudate may exist as a single form or in combinations (e.g., serosanguineous):

- Serous or clear fluid
- Sanguineous for blood
- Purulent pus made up of inflammatory cells and tissue debris that can result from infection or an inflammatory

process

Amount

The amount of exudate may indicate that the cause of the wound has not been treated (e.g., edema due to venous insufficiency), that congestive heart failure is present (look for bilateral involvement and extension above the knee), that low albumin levels have occurred (malnutrition, kidney or liver disease), or that infection is present (check for signs or symptoms). Amounts of exudate include:

- none
- small—there's just a detectable discharge when the dressing is removed, less than 33%
- moderate—exudate is covering less than 67% of the dressing surface
- large—exudate is covering more than 67% of the dressing surface.

Exudate may be serous (clear or pale yellow), serosanguineous (serous or blood tinged), sanguineous (bloody), or greenish-brown. The consistency may be thick, milky, or purulent. It can be an important indicator of wound status (Fig. 6-8).

Figure 6-8. Venous insufficiency ulcer with necrotic tissue liquefaction, fibrin, and slough. Courtesy of Jeff Niezgoda, used with permission.

Sepsis

Sepsis or bacteremia is caused by anaerobes and gram-negative bacteria and can occur in any susceptible wound. Assessment for the presence of sepsis should include consideration of erythema, warmth, edema, purulent or increased drainage, induration, increased tenderness or pain, and crepitus or fluctuance.[3,39–41] Consider local infection in a pressure injury in the presence of nonhealing for 2 weeks, friable granulation tissue, malodor, increased pain or heat in the wound or periwound, increased drainage, an ominous change in the nature of wound drainage, an increase in necrotic tissue in the

wound bed, and pocketing or bridging in the wound bed.[3] Also be suspicious of infection if the wound is located in an area likely to be contaminated (e.g., anus), in diabetes mellitus, protein–calorie malnutrition, hypoxia or poor tissue perfusion, immune-compromised individuals, or those with autoimmune diseases.[3] If sepsis is present, it's important to determine whether the infection is local or systemic. Interventions are based on accurate assessment and laboratory support. Infrared thermography using a noncontact thermometer can be used to assess for infection on the feet of persons at risk for or with diabetic foot ulcers.[19,42,43]

The best method to culture a wound to determine the presence of organisms remains controversial. Typically, tissue biopsy and quantitative culture are the gold standard.[3] These options may not be available in all settings, however, and many clinicians lack the skill necessary to perform them. The swab method, in which the wound must be cleaned and thoroughly dried prior to swabbing for a culture, continues to be used in many settings. After first cleansing the wound, culture the viable tissue in the wound bed to identify the presence and type of microorganisms.[39] Culturing of wounds is important in determining whether infection is present and, if so, identifying the microorganism(s). Malodorous wounds should also be documented. However, make sure the odor is from the wound—not the dressing change—which is a common mistake. The clinician must first cleanse the wound prior to assessing and documenting malodor. Certain organisms—such as *Pseudomonas*—have a distinct odor that is easily recognized by the trained clinician[40,41] (see Chapter 7, Wound Bioburden and Infection, for more detailed information on infection and culturing).

Surrounding Skin/Periwound/Maceration

The periwound is the skin surrounding a wound, and it also provides valuable information to the assessing clinician. Erythema and warmth may indicate inflammation, cellulitis (Fig. 6-9A and B), or infection. Interruptions in periwound skin integrity (denudation, erosion, papules, or pustules) may indicate allergic reactions to tape or dressing adhesive. Moisture-associated skin damage (MASD) caused

by wound drainage can lead to maceration of the periwound skin. Maceration or desiccation may be a sign that the dressing is too moist or too dry for the amount or type of exudate. Ensure that the dressing used can adequately absorb the amount of drainage present without having to be changed frequently. Palpation should be done with the fingertips around a wound surface. This may reveal induration (hard to touch) or fluctuance (bubbly, fluid wave), which are abnormal fluid accumulations indicative of further tissue damage or abscess. In a study of lower extremity venous ulcers, more rapid closure of the wound bed occurred when treatments were also applied to the periwound area. Although research provides us with a different understanding of where the wound ends[44] and the surrounding skin begins, assessment of surrounding tissue does provide useful information for the ongoing evaluation and future wound care interventions.

Figure 6-9. (A) Maceration. This photograph shows maceration of the surrounding skin caused by an overwhelmed dressing. **(B)** Neuropathic ulcer with periwound callous and maceration noted. Courtesy of Jeff Niezgoda, used with permission.

Practice Point

Cleanse the wound prior to assessing for malodor. A change in the odor of a wound must raise the index of suspicion for the onset or presence of wound infection. However, remember that not all odor indicates infection; certain dressings develop a distinct odor when

exudates interact with them (e.g., alginates). Odor may also indicate the need to change the dressing more often.

As stated, MASD from wound exudate may cause maceration of the periwound skin. Maceration is a softening of the skin surrounding a wound caused by excess drainage or pooling of fluid on intact skin and appears as a white, waterlogged area. It may be caused by inadequate management of exudate or an increase in exudate due to changes in the wound tissue. Maceration may be prevented by using an appropriate barrier product (liquid acrylate, cream, ointment, etc.) around the wound, changing the dressing more often, or selecting a more absorbent dressing (Fig. 6-9A and B).

Edges and Epithelialization

Epithelialization is the regeneration of the epidermis across a wound surface.[36] The epithelial wound edge is continuous and often difficult to see. As wound migration proceeds from the edges toward the center, the portion covered with epithelium appears pearly or silver and shiny. Because it is thin and fragile, this area is easily damaged. The edge of a wound may be attached to the wound bed, unattached (undermining), or rolled inward (Figs. 6-10 and 6-11). Wound edges should be assessed as part of a thorough evaluation of the wound.

Figure 6-10. Wound edges with epithelialization. In this photograph, wound edges are attached and epithelialization is present.

Figure 6-11. Rolled edges. Wound edges rolled inward.

Examining the wound edges may reveal whether the wound is acute or chronic and can often provide clues as to the wound's etiology. For example, a wound with inflamed edges or violaceous with undermined borders may indicate pyoderma gangrenosum. A wound with edges rolled inward may be too dry, causing the wound edges to seek more moisture from the wound bed. A wound that is covered in necrotic tissue, desiccated, or deprived of oxygenation will exhibit poorly defined wound margins.[37]

Epithelialization can also occur in the middle of a wound bed if hair follicles or new cell growth is present. The appearance of new tissue at the wound edge can be measured in centimeters or by the percentage of wound coverage (e.g., "0.3 cm of epithelial tissue surrounds the wound" or "wound is 25% epithelialized"). The degree of epithelialization is often overlooked.

Necrotic Tissue

Necrotic tissue is a dead, devitalized, avascular tissue that provides an ideal medium for bacterial proliferation and may inhibit healing. It's a well-known theory that wound healing is optimized when all necrotic tissue is removed from the wound bed. Necrotic tissue may present as yellow, gray, brown, or black. As it becomes dry, it presents as thick, hard, leathery black eschar.[37] Yellow, soft moist avascular (devitalized) tissue is referred to as slough.[37] It may be loose or firmly adherent as seen in Figure 6-12. Document the type and percent of necrotic tissue in the wound bed. For example, the wound bed may be 100% necrotic or 25% granular with 75% necrotic tissue. Slough is not present in stage 1 and 2 pressure injuries. Biofilm can also be found in chronic wounds. A biofilm is "an aggregate of microorganisms known to cause chronic inflammation … they have enhanced resistance to destruction by endogenous antibodies and phagocytic cells, as well as by exogenous antibiotics and antiseptics.

Biofilms play an important role in maintaining a chronic inflammation state ultimately leading to the failure to heal of skin wounds."[3,45]

Figure 6-12. Leg ulcer with drainage, fibrin, and slough firmly attached. Note also periwound inflammation. Courtesy of Jeff Niezgoda, used with permission.

Wound Bed Tissue

The wound bed tissue reveals the phase and progress of wound healing through observation of its color, degree of moisture—a moist wound bed facilitates movement of fibroblasts and macrophages, as

well as collagenase, and other chemicals, across the wound bed, resulting in healing—and amount of epithelialization.[44,46] The wound bed may be pale pink, pink, red, yellow, or black. Clean, granular wounds are typically described as red, and wounds with devitalized slough are described as yellow. Brown and black wounds are typically those with necrotic tissue or eschar or desiccated tissue; these wounds need to be debrided because this type of tissue slows the healing process.[37]

Is the wound bed moist or dry? The presence of moisture or dry tissue will guide you in selecting the right dressing to create an environment that supports healing. Do you see new tissue growth—epithelialization at the wound edges or within the wound bed? Is granulation tissue present—that is, beefy red tissue with a granular or gritty appearance?

Documentation should be based on your observations.[47] Is the wound 100% granular tissue, or is it 25% filled with slough (yellow tissue) or necrotic (dead) tissue? All three tissue types can be found in the same wound, and assessing the amount of each type of tissue will help you determine the appropriate treatment and document the outcome of care based on improvement or deterioration, as indicated by wound tissue characteristics. Outcomes can then be tracked by percentage of improvement toward a clean granular wound bed (e.g., “the wound progressed from 75% necrotic tissue to 100% granular tissue”).

Tenderness to touch and the amount of pain the patient reports—both in the wound itself and in the surrounding tissue—are also essential parts of your assessment. Wound pain is one of the secondary signs of infection. It’s important to differentiate between constant and episodic pain (such as pain that occurs only with dressing changes). Use a validated pain assessment scale accepted by your facility[3] (see Chapter 7, Wound Bioburden and Infection, and Chapter 12, Pain Management and Wounds).

A variety of proteins are present in the wound bed. In a longitudinal study, a difference in 21 levels of wound proteins in different areas of wound tissue (peripheral vs. internal) was identified between chronic pressure injuries and those that healed.[48]

Assessing and Measuring Healing

Although wound assessment and measurement are important, so is documenting wound healing. There is growing research on the best ways to determine the healing rates of different types of chronic wounds (pressure, venous, and diabetic neuropathic ulcers) as well as expected healing rates at 4 weeks.[49,50] The 2019 International Pressure Injury Prevention and Treatment Guideline states "pressure injuries receiving appropriate care will demonstrate signs of healing within 2 weeks."[3] A variety of tools are available to assess and document healing in clinical practice, including the Pressure Sore Status Tool (PSST),[51,52] the Pressure Ulcer Scale for Healing (PUSH),[53] the Toronto Symptom Assessment System for Wounds (TSAS-W),[54] and the DESIGN-R from Japan.[55]

Originally developed for pressure injuries, the PSST has been revised by the author, Dr. Barbara Bates-Jensen, to be used for all wounds and is now referred to as the Bates-Jensen Wound Assessment Tool (BWAT).[56] The BWAT includes 13 wound factors to be tracked over time, each of which is scored numerically. Wound location and shape are not scored. The total of the 13 factors reflects overall wound status.[56]

The PUSH tool, developed and revised by the NPIAP,[53] has been validated by research. This tool allows for the quick and reliable assessments necessary to monitor pressure injury healing over time and should be used at least weekly.[53,57] Three scores are developed: one is for surface area (L × W), one indicates drainage amount, and the third is for tissue type. The total score, which is the sum of these three factors, is plotted on a healing record (or graph) to depict healing over time. Although originally developed by the NPIAP to quantify healing in pressure injuries, a study by Hon and colleagues[58] provides evidence that this tool is valid for monitoring and evaluating the progress of venous and diabetic ulcers.

The TSAS-W, which can be completed by either the patient or caregiver,[54] quantifies 10 wound-related symptoms. The patient or the

caregiver ranks each symptom from 0 to 10. In a test on 531 patients, this tool was found to be useful for all wound types.

Another tool is the DESIGN from Japan, which is used to classify severity and monitor healing of pressure injuries.[59] The tool uses six factors to classify and assess healing: *d*epth, *e*xudate, *s*ize, *i*nfection, *g*ranulation, and *n*ecrosis. Reported interrater reliability is good with both patients and photos, and there is very good correlation with the BWAT.[59]

Wound Documentation Essentials

Documentation is an essential component of wound assessment. Every wound assessment should be documented thoroughly, accurately, and legibly, with an accompanying signature as well as the date and time of the assessment. Wounds should be documented on the patient's admission, weekly, with each dressing change, upon any significant change in the wound, and upon discharge.

As mentioned earlier, the initial assessment and documentation become a baseline comparison for all future assessments. It is recommended that each clinical agency have a consistent chart form and format for wound documentation. All facilities should follow the wound assessment policy as determined by their setting-specific regulations, but to identify ulcers/wounds present on admission, assessment and documentation should take place at least within the first 24 hours.

These include the mandated Outcome and Assessment Information Set D (OASIS D)[60] for home care or the MDS 3.0 for long-term care.[13]

Practice Point

It's important to remember that in the United States, the OASIS and MDS are regulatory documentation tools—neither tool is considered a comprehensive wound assessment.

Wound Photography

Photographs can provide a visual record of the wound. When done correctly, they can assist the clinician in clinical care decisions and provide documentation support in the case of litigation[61] but should not replace bedside assessments. Standardization of when and how to photograph wounds is paramount. It is important to remember, however, that photographs do not replace bedside wound assessments.

When using photography, it's necessary to permanently mark on the photograph the date and time it was taken as well as patient identification information. The photo should also include a sample measure in each frame, such as a 10-cm strip of paper tape. For the best clarity when using digital photography, a camera no smaller than 1.5 megapixels is recommended. However, because digital photos can be altered, the identifying data must be encoded permanently. When periodic photos are taken, distortion of the photos can be a problem due to body contour, angle of the body in a bed, angle and distance of the camera from the patient, and lighting. Some photo software packages use a target plate in the photo and the software; once the photo is downloaded, it can automatically calculate wound area and volume as well as length and width. Wound photographs can quickly and accurately represent a wound's appearance if the proper equipment is used in the correct way.[62,63] Clinicians need appropriate training and must consistently follow a wound photography protocol. The angle at which the camera is held to the wound makes a difference. Using the wrong camera angle can result in an improperly photographed wound that appears larger or more or less deteriorated than it truly is (Fig. 6-13). Other strategies to enhance wound photos have been described elsewhere in the literature.[28,64]

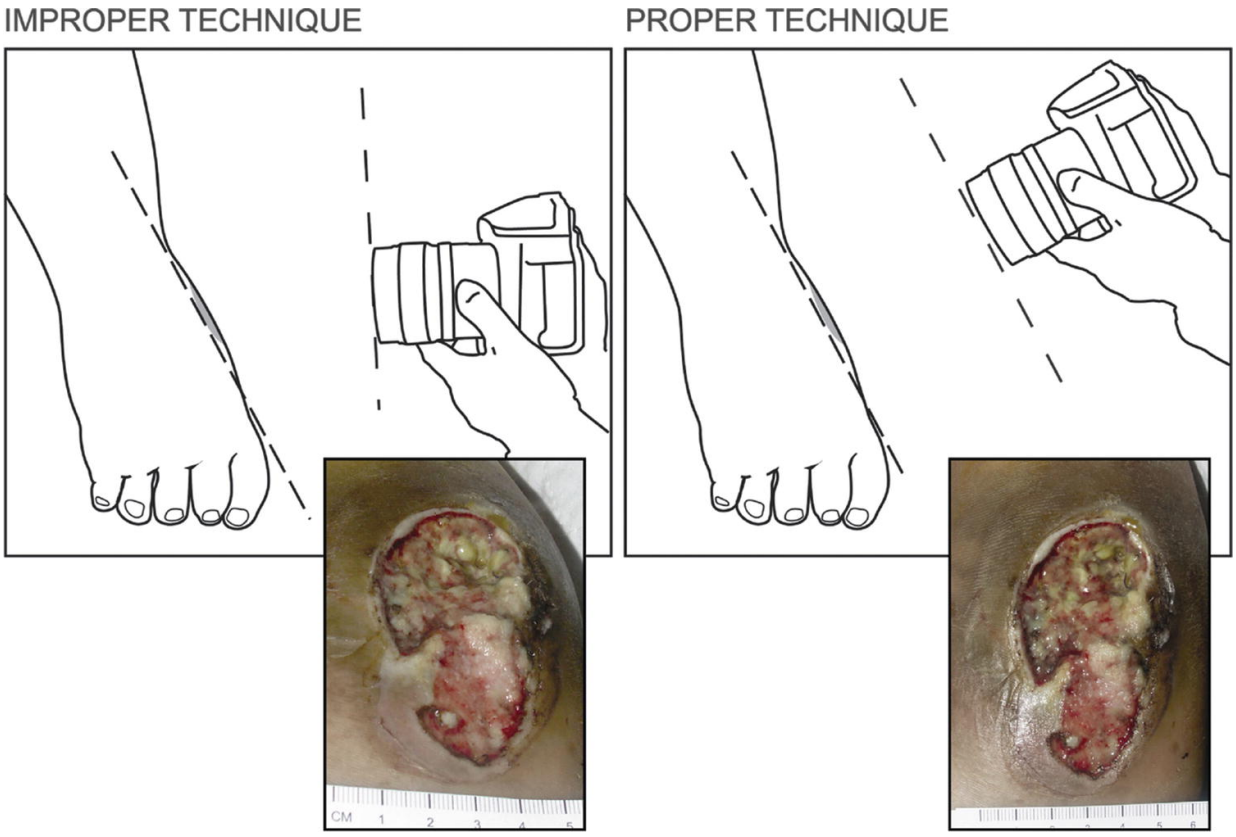


Figure 6-13. Proper photo technique. Used correctly, photography can be a great tool in documenting the appearance of a wound. Holding the camera at the appropriate angle is imperative to accurately document wound size. (Reprinted with permission from Rennert, R., Golinko, M., Kaplan, D., et al. "Standardization of Wound Photograph Using the Wound Electronic Medical Record." *Advances in Skin & Wound Care* 22(1):32-8, 2009. Copyright © 2009 Lippincott, Williams and Wilkins.)

Wound photography continues to be significantly impacted by advances in technology. Basic capture of a digital image is no longer the gold standard. Digital imaging now provides a variety of enhancements to compliment wound assessment. As mentioned

earlier, digital imaging can provide wound measurements including the area, depth, and volume. Technologies are being introduced that can assess the wound characteristics such as the percentage of granulation and fibrin and necrotic tissue present on the surface. Digital software can provide analysis of sequential wound dimensions to give analysis and graphic presentations of changes in healing trajectories. Sophisticated imaging devices are available that measure wound perfusion and oxygenation, which can be used to monitor responses to therapy, such as debridement and hyperbaric oxygen.[65] While many of these technologies are still being vetted for clinical applicability, and reimbursement issues must be resolved, the wound care specialist must be aware of the technologies that are available to potentially enhance care and improve patient outcomes.

Electronic Medical Records and Electronic Health Records

The EMR is the legal record created in hospitals and healthcare environments that is the source of data for the electronic health record (EHR). The EHR represents the ability to easily share medical information among stakeholders and to have a patient's information follow him or her through the various modalities of care in which he or she engages. EHRs are reliant on EMRs being in place, and EMRs will never reach their full potential without interoperable EHRs in place.[66]

A system of dedicated wound EMRs supports comprehensive and consistent care in patients with wounds. It promotes patient safety, facilitates evidence-based care,[63] and helps eliminate disparities in care regardless of care setting because the interdisciplinary team has access to objective wound assessment data.[2,16,17] In one large urban center, the use of an objective wound EMR decreased chronic wound emergencies.[2]

Telemedicine

Teleassessment and telemanagement are terms used to describe the "assessment and management of a wound at a distance using electronic information and communication technologies."[28] Telemedicine can enhance wound treatment in the home, at outlying clinics, and in inclement weather when travel is impossible. In one randomized controlled study of 103 participants with 160 pressure injuries or nonhealing surgical wounds receiving home care in conjunction with telemedicine, the average healing or improvement rate was 51 days for pressure injuries and 34 days for surgical wounds.[67] The role of telemedicine in wound healing continues to evolve. In another descriptive comparative study of home care nurses with 43 adult patients, the use of digital photography rather than simply reporting resulted in a decreased chance of under- or overtreating the patients' wounds.[68] Use of digital images in telemedicine also resulted in the unexpected finding of more information than just what was revealed by conventional inspection, such as the identification of other factors in the patient's environment beyond the local wound bed that may have inhibited wound healing.[68] These included, for example, the patient's bed, wheelchair, redistribution devices, and shoes.[68] In areas of the country where weather can prohibit travel to a wound care center or the patient is unable to be transported, telemedicine has been an immense facilitator of timely and appropriate wound assessment and treatment. Truly this was an example of the importance of treating the "whole patient and not just the hole in the patient."

Summary

Wound assessment—an appraisal of a patient's condition based on clinical signs and symptoms, laboratory data, and medical history—is an integral part of wound management. Assessment has become a highly specialized area of care, requiring well-developed observational skills and current knowledge. The use of current terminology is vital to accurate assessment and communication. Use of the Pressure Injury and Wound ASSESSMENTS[23] chart or WOUND PICTURE[47] mnemonic can provide a fast, ongoing, and accurate assessment for patients with wounds[50] (Box 6-4). Other tools that quantify wound characteristics, such as the BWAT,[56] PUSH,[53] TSAS-W,[54] DESIGN,[59] or DESIGN-R,[55] can help the clinician track wound healing. Proper assessment of wound parameters provides clinicians with the information they need to make the decisions that guide the wound care team to suitable interventions, management, and care strategies. Technology will continue to help clinicians with documentation. Unless standardized techniques and protocols are followed, wound photos can hinder rather than help with treatment care decisions. EMRs and their important role in EHRs are the wave of the future. These advances, coupled with the use of telemedicine, ensure that the future of wound assessment and documentation looks promising.

Box 6-4 Wound Picture

When assessing wounds in your patient, another helpful mnemonic is, **WOUND PICTURE**, for a fast and accurate assessment.

Wound or ulcer location

Odor Assess before and during all dressing changes

Ulcer category, stage (for pressure injury) or classification (for diabetic ulcer), and depth (partial thickness or full thickness)

Necrotic tissue

Dimension of wound (shape, length, width, depth); drainage color, consistency, and amount (scant, moderate, large)

Pain (when it occurs, what relieves it, patient's description, patient's rating on scale of 0 to 10)

Induration (surrounding tissue hard or soft)

Color of wound bed (red–yellow–black or combination)

Tunneling (record length and direction—toward patient's right, left, head, feet)

Undermining (record length and direction, using clock references to describe)

Redness or other discoloration in the surrounding skin

Edge of skin loose or tightly adhered? Edges flat or rolled under

From *Wound Care Made Incredibly Easy*, 3rd ed. Philadelphia, PA: Lippincott, Williams & Wilkins, 2015.

PATIENT SCENARIO

Clinical Data

Mr. KZ is a 67-year-old gentleman with a painful stage 4 pressure injury on the right trochanter. There is a large amount of light beige exudate that has some blood in it. There is a small amount of black eschar in the wound bed, while the rest of the wound bed is red and granular.

Case Discussion

After the nurse removes the dressings and cleanses the pressure injury, he or she completes the pressure injury and wound assessment chart documentation (see Box 6-2).

Wound Etiology: Pressure Injury

Anatomic location: right trochanter; date of onset: chronic, 6 weeks ago

Stage: 4; size: 7.5 × 6.2 × 2.5 cm (L × W × D); shape: irregular

Sinus tract/tunneling presents with undermining: 0.75 cm from 1 o'clock to 4 o'clock

Exudate: large amount, thin, tannish/serosanguineous; malodorous drainage

Sepsis: local

Surrounding skin: erythematous, intact, and slightly swollen; temperature: skin warm to touch

Maceration: not present

Edges/epithelialization, attached and slightly rolled; epithelialization, small amount present, 0.05 cm

Necrotic tissue: present; black, less than 10%

Tissue wound bed, granulation, present 90%; tenderness to touch, pain present; anytime; pain scale 9/10

Status: initial assessment date, 4/23/19; nutritional, PT consult ordered 4/24/19

Signature: Good Nurse RN

Show What You Know

1. **Initial wound assessment involves all of the following except:**
    A. observation.
    B. data collection.
    C. evaluation.
    D. surgical debridement.

2. **A wound that has tissue damage through the epidermis and partially into the dermis would be classified as:**
    A. superficial.
    B. partial thickness.
    C. full thickness.
    D. subdermal.

3. **In assessing a wound, you find an area of tissue destruction under the edge of the patient's wound. This is best described as:**
    A. a sinus tract.
    B. maceration.
    C. fistula.
    D. undermining.

4. **Which of the following wound photography techniques is *incorrect*?**
    A. Include a measuring device in the photo.
    B. Permanently mark the date and time of the photo in the picture.
    C. Hold the camera at a 90-degree angle to the wound.
    D. Digital photos should be at least 1.5 megapixels.

5. **Which of the following statements about electronic health records is <u>false</u>? They are:**
    A. the same as electronic medical records.
    B. reliant on electronic medical records being in place.

C. an important way of sharing medical information across stakeholders.
D. a modality of care that holds promise to promote patient safety across care settings.

Answers to these questions are found on page 682.

References

1. Bates-Jensen, B.M. Assessment of the Patient with a Wound. In D. Doughty, L. McNichols, eds. Wound Ostomy and Continence Nurses's Society Core Curriculum, Wound Management. Philadelphia, PA: Wolters Kluwer, 2016:38-67.
2. Golinko, M.S., Clark, S., Rennert, R., et al. "Wound Emergencies: The Importance of Assessment, Documentation and Early Treatment using a Wound Electronic Medical Record." Ostomy/Wound Management 55(5):54-61, 2009.
3. European Pressure Ulcer Advisory Panel, National Pressure Injury Advisory Panel and Pan Pacific Pressure Injury Alliance. In: E. Haesler, ed. Prevention and Treatment of Pressure Ulcers/Injuries: Clinical Practice Guideline. The International Guideline. EPUAP/NPIAP/PPPIA, 2019.
4. Larazus, G.S., et al. "Definitions and Guidelines for Assessment of Wounds and Evaluation of Healing." Archives of Dermatology 130(4):489-93, April 1994.
5. Centers for Medicare and Medicaid Services (CMS). "Usual Care in the Management of Chronic Wounds: A review of recent literature." Retrieved April 24, 2019 from http://www.cms.gov/Medicare/Coverage/DeterminationProcess/downloads/id37TA.pdf
6. Bower, MG. "Evaluating and Managing Bite Wounds." Advances in Skin & Wound Care 15(2):88-90, March-April 2002.
7. Sibbald, R.G., Goodman, L., Woo, K.Y., et al. "Special Considerations in Wound Bed Preparation 2011: An Update©." Advances in Skin & Wound Care 24(9):415-36, quiz 436-8, 2011.
8. Rice, J. Wound Care Manual and Clinical Guidelines for Nurses, Feb. 2017. Retrieved April 24, 2019 from https://www.ausmed.com/cpd/articles/wound-care-assessment
9. Sprigle, S., Zhang, L., Duckworth, M. "Detection of Skin Erythema in Darkly Pigmented Skin using Multispectral Images." Advances in Skin & Wound Care 22(4):172-79, 2009.
10. Okan, D., Woo, K., Ayello, E.A., et al. "The Role of Moisture Balance in Wound Healing." Advances in Skin & Wound Care 20(1):39-53, 2007.
11. Conley, M. "Wound, Ostomy, Continence Nurses Society Pressure Ulcer Evaluation: Best Practice for Clinicians." WOCN Society 9, 2008.
12. Centers for Medicare and Medicaid Services (CMS). "Pressure Ulcers, Revised Guidance for Surveyors in Long Term Care." Tag F-314, issued November 12, 2004. Retrieved June 11, 2006 from http://new.cms.hhs.gov/manuals/downloads/som107ap_pp_guidelines_ltcf.pdf. Last accessed 13 June 2019.

13. Centers for Medicare and Medicaid Services (CMS). State Operations Manual. Guidance to Surveyors. F 686. October 2018. Available at https://www.cms.gov/Regulations-and-Guidance/Guidance/Manuals/downloads/som107ap_pp_guidelines_ltcf.pdf
14. Banfield, K.R., Shuttleworth, E. "A Systematic Approach with Lasting Benefits: Designing and Implementing a Wound Assessment Chart." Professional Nurse 8(4):234-8, January 1993.
15. Mazuz, R. "4 Ways to Integrate Digital Health into the Wound Center Today." Today's Wound Clinic 13(4):9-10, November 2018.
16. Howell, R.S., Gorenstein, S., Gillette, B.M., et al. "A Framework to Assist Providers in the Management of Patients with Chronic Nonhealing Wounds." Advances in Skin & Wound Care 31(11):491-500, November 2018.
17. Howell, R.S., Kohan, L.S., Woods, J.S., et al. Wound Care Center of Excellence: A Process for Continuous Monitoring and Improvement of Wound Care Quality. Advances in Skin & Wound Care 31(5):204-13, May 2018.
18. Persaud, R., Coutts, P.M., Brandon, A., et al. Validation of the Healthy Foot Screen: A Novel Assessment Tool for Common Clinical Abnormalities. Advances in Skin & Wound Care 31(4):154-62, April 2018.
19. Sibbald, R.G., Ayello, E.A., Alavi, A., et al. Screening for the High-Risk Diabetic Foot: A 60-Second Tool (2012). Advances in Skin & Wound Care 25(10): 465-76, October 2012.
20. Woodbury, M.G., Sibbald, R.G., Ostrow, B., et al. Tool for Rapid & Easy Identification of High Risk Diabetic Foot: Validation & Clinical Pilot of the Simplified 60 Second Diabetic Foot Screening Tool. PLoS One 10(6):e0125578, 2015.
21. "Burn Percentage in Adults: Rule of Nines." Retrieved September 30, 2010 from www.emedicinehealth.com/burn_percentage_in_adults_rule_of_nines/article_em.htm
22. The Royal Children's Hospital Melbourne. "Clinical Practice Guidelines: Burns." Retrieved May 26, 2010 from www.rch.org.au/clinicalguide/cpg.cfm?doc_id=5158
23. Ayello, E. "Teaching the Assessment of Patients with Pressure Ulcers." Decubitus 5(7):53-4, July 1992.
24. Centers for Medicare and Medicaid Services. "Nursing Home Quality Initiatives: MDS 3.0 Overview." Retrieved April 29, 2019 from http://www.cms.hhs.gov/NursingHomeQualityInitatives
25. Levine, J.M., Roberson, S., Ayello, E.A. "Essentials of MDS 3.0, Section M, Skin Condition." Advances in Skin & Wound Care 23(6):273-84, quiz 285-6, 2010.
26. National Pressure Injury Advisory Panel (NPIAP). The Facts About Reverse Staging in 2000. The NPUAP Position Statement. Available at

http://www.npuap.org/wp-content/uploads/2012/01/Reverse-Staging-Position-Statement.pdf. Last accessed 13 August 2014.
27. NPUAP Pressure Injury Staging System Changes. April, 2016. Available at https://www.npuap.org/resources/educational-and-clinical-resources/npuap-pressure-injury-stages/. Last accessed 13 June 2019.
28. Ahn, C., Salcido, R.S. “Advances in Wound Photography and Assessment Methods.” Advances in Skin & Wound Care 21(2):85-93, 2008.
29. Langemo, D., Anderson, J., Hanson, D., et al. “Measuring Wound Length, Width and Area: Which Technique?” Advances in Skin & Wound Care 21(1): 42-5, quiz 46-7, 2008.
30. Langemo, D.K., Melland, H., Hanson, D., et al. “Two-dimensional Wound Measurement: Comparison of 4 Techniques.” Advances in Skin & Wound Care 11(7): 337-43, 1998.
31. Langemo, D.K., Melland, H., Olson, B., et al. “Comparison of 2 Wound Volume Measurement Methods.” Advances in Skin & Wound Care 14(4):190-6, 2001.
32. Langemo, D., Spahn, J., Spahn, T., et al. “Comparison of Standardized Clinical Evaluation of Wounds Using Rule Length by Width and Scout Length by Width Measure and Scout Perimeter Trace.” Advances in Skin & Wound Care 28(3):116-21, 2015.
33. Langemo, D., Spahn, J., Snodgrass, L. “Accuracy and Reliability of the Wound Shape Measuring and Monitoring System.” Advances in Skin & Wound Care 28(7):317-23, 2015.
34. Kieser, D.C., Hammond, C. Leading Wound Care Technology: The ARANZ Medical Silhouette. Advances in Skin & Wound Care 24(2):68-70, February 2011.
35. Frantz, R.A., Johnson, D.A. “Stereophotogrammetry and Computerized Image Analysis: A Three-dimensional Method of Measuring Wound Healing.” Wounds 4:58-64, 1992.
36. Kundin, J.I. “A New Way to Size Up a Wound.” American Journal of Nursing 1:206-7, 1989.
37. Bryant, R.A., Nix, N.P. Acute and Chronic Wounds, Current Management Concepts, 3rd ed. St. Louis, MO: Mosby Year Book, 2007.
38. Ayello, E.A., Sibbald, R.G. “LOWE© Skin Barriers for Wound Margins: 20 Second Enablers for Practice.” Advances in Skin & Wound Care 19(5):237, 2006.
39. Harding, K.A., Carville, K., Cuddigan, J., et al. “Wound Infection in Clinical Practice: Shaping the Future. An International Consensus Document.” International Wound Journal 5(Suppl 3):1-11, 2008.
40. Mouton, C.P., Bazaldua, O.V., Pierce, B., et al. “Common infections in Older Adults.” American Family Physician 63(2):257-68, 2001.
41. Gardner, S.E., Frantz, R.A., Doebbeling, B.N. “The Validity of the Clinical Signs and Symptoms Used to Identify Localized Chronic Wound Infection.”

Wound Repair and Regeneration 9(3):178-86, May-June 2001.
42. Sibbald, R.G., Mufti, A., Armstrong, D.G. Infrared Skin Thermometry: An Underutilized Cost-effective Tool for Routine Wound Care Practice and Patient High-Risk Diabetic Foot Self-monitoring. Advances in Skin & Wound Care 28(1):37-44, January 2015.
43. Mufti, A., Sibbald, R.G. Validation of Commercially Available Infrared Thermometers for Measuring Skin Surface Temperature Associated with Deep and Surrounding Wound Infection. Advances in Skin & Wound Care 28(1):11-6, January 2015.
44. Stojadinovic, O., et al. “Role of B-Catenin and C-myc in the Inhibition of Epithelialization and Wound Healing.” American Journal of Pathology 167(1):59-69, 2005.
45. James, G.A., Swogger, E., Wolcott, R., et al. “Biofilms in Chronic Wounds.” Wound Repair and Regeneration 16(1):37-44, 2008.
46. Maklebust, J., Sieggreen, M. Pressure Ulcers: Guidelines for Prevention and Management, 3rd ed. Springhouse, PA: Springhouse Corp., 2000.
47. Wound Care Made Incredibly Easy, 2nd ed. Philadelphia, PA: Lippincott, Williams & Wilkins, 2006.
48. Edsberg, L.E., Wiffels, J.T., Ha, D.S. “Longitudinal Study of Stage III and Stage IV Pressure Ulcer Area and Perimeter as Healing Parameters to Predict Wound Closure.” Ostomy/Wound Management 57(10):50-62, 2011.
49. Jessup, R.L. “What Is the Best Method for Assessing the Rate of Wound Healing? A Comparison of 3 Mathematical Formulas.” Advances in Skin & Wound Care 19(3):138, 140-2, 145-6, 2006.
50. Van Rijswijk, L. “Full-thickness Pressure Ulcers: Patient and Wound Healing Characteristics.” Decubitus 6(1):16-21, 1993.
51. Bates-Jensen, B. “The Pressure Sore Status Tool a Few Thousand Assessments Later.” Advances in Wound Care 10(5):65-73, 1997.
52. Bates-Jensen, B.M. “A Quantitative Analysis of Wound Characteristics as Early Predictors of Healing in Pressure Sores” [Abstract]. Dissertation Abstracts International, 59:11. Los Angeles, CA: University of California, Los Angeles, 1999.
53. National Pressure Ulcer Advisory Panel. “PUSH Tool.” Retrieved April 15, 2010 from http://www.npuap.org/PDF/push3.pdf
54. Maida, V., Ennis, M., Kuziemsky, C. “The Toronto Symptom Assessment System for Wounds: A New Clinical and Research Tool.” Advances in Skin & Wound Care 22(10):468-74, 2009.
55. Lizaka, S., Kaitani, T., Sugama, J., et al. “Predictive Validity of Granulation Tissue Color Measured by Digital Image Analysis for Deep Pressure injury Healing: A Multicenter Prospective Cohort Study.” Wound Repair and Regeneration 21(1):25-34, 2013.
56. Harris, C., Bates-Jensen, B., Parslow, N., et al. “Bates-Jensen Wound Assessment Tool, Pictorial Guide Validation Project.” Journal of Wound,

Ostomy, and Continence Nursing 37(3):253-9, May-June 2010.
57. Stotts, N.A., et al. “Testing the Pressure Ulcer Scale for Healing (PUSH) and Variations of the PUSH.” Paper presented at the 11th Annual Symposium on Advanced Wound Care, April 18–22, 1998, Miami Beach, FL.
58. Hon, J., Lagden, K., McLAren, A., et al. “A Prospective, Multicenter Study to validate use of the Pressure Ulcer Scale for Healing (PUSH©) in Patients with Diabetic, Venous and Pressure injuries.” Ostomy/Wound Management 56(2):26-36, 2010.
59. Sanada, H., Moriguchi, T., Miyachi, Y., et al. “Reliability and Validity of DESIGN, a Tool that Classifies Pressure Ulcer Severity and Monitors Healing.” Journal of Wound Care 13(1):13-8, 2004.
60. Centers for Medicare and Medicaid Services. Home Health Quality Reporting Program. Available at https://www.cms.gov/Medicare/Quality-Initiatives-Patient-Assessment-Instruments/HomeHealthQualityInits/index.html. Accessed April 29, 2019.
61. International Expert Wound Care Advisory Panel; Ayello, E.A., Capitulo, K.L., Fife, C.E., et al. “Legal Issues in the Care of Pressure Ulcer Patients: Key Concepts for the Healthcare Providers: A Consensus Paper from the International Expert Wound Care Advisory Panel.” WCET Journal 29(3):8-22, 2009.
62. Sullivan, V. “In Focus: The Photography Forecast.” Today’s Wound Clinic 2(2):30-1, 33, 2008.
63. Rennert, R., Golinko, M., Kaplan, D., et al. “Standardization of Wound Photograph using the Wound Electronic Medical Record.” Advances in Skin & Wound Care 22(1):32-8, 2009.
64. Rennert, R., Golinko, M., Yan, A., et al. “Developing and Evaluating Outcomes of an Evidence-based Protocol for the Treatment of Osteomyelitis in Stage IV Pressure Ulcers: A Literature and Wound Electronic Medical Record Database Review.” Ostomy/Wound Management 55(3):42-53, 2009.
65. Bowen, R.E., Treadwell, G.R.N., Goodwin, M.R.R.T. Correlation of Near Infrared Spectroscopy Measurements of Tissue Oxygen Saturation with Transcutaneous pO2 in Patients with Chronic Wounds. SM Vascular Medicine 1(2):1006, 2016.
66. Garets, D., David, M. “Electronic Medical Records vs. Electronic Health Records: Yes, There is a Difference. A HIMSS Analytics™ White Paper. Updated January 26, 2006.” Retrieved May 26, 2010 from www.himssanalytics.org
67. Terry, M., Halstead, L.S., O’Hare, P., et al. “Feasibility Study of Home Care Wound Management using Telemedicine.” Advances in Skin & Wound Care 22(8):358-64, 2009.
68. Buckley, K.M., Adelson, L.K., Agazio, J.G. “Reducing the Risks of Wound Consultation: Adding Digital Images to Verbal Reports.” Journal of Wound,

Ostomy, and Continence Nursing 36(2):163-70, March-April 2009. doi: 10.1097/01.2009.

7

Wound Bioburden and Infection

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Objectives

After completing this chapter, you'll be able to:

- compare and contrast the various phases of the wound bioburden cycle (contamination, colonization, critical colonization/biofilm, and infection)
- propose how to identify infection using signs and symptoms and diagnostic tools
- describe biofilm, how it is formed and its role in wound healing
- propose treatment for increased bioburden, biofilm, and infection.

Bioburden in Wounds

Microorganisms and man normally live in harmony.[1] Gene sequencing of the skin microbiome has shown that organisms living on the skin are associated with moist, dry, and oil microenvironments.[2] In addition, the microorganisms at one wound site are not necessarily like those at another site on the same person, nor is the microbiome of one person like that of another.

Practice Point

The microbes that inhabit one body site may not be the same as found in another site on the same person.

The skin is a mechanical barrier to organisms, and skin oils and sweat are chemicals that help prevent their entrance into tissue.[1] When a wound occurs, organisms from the skin, gastrointestinal track, and mucous membranes as well as the external environment have free access to tissue. Bioburden is a measure of the number of viable microbes on a tissue surface or device.

Practice Point

Bioburden is a measure of the viable organisms in a wound or on a tissue surface.

Until recently, scientists and clinicians understood that infection was due to planktonic organisms, those who float free when placed in water.[3] These organisms are individual cells that have a predictable life cycle and work independently. There is a good understanding of planktonic organisms, as seen by the fact that antibiotics have been developed that target specific sites to kill or immobilize them. Our growing understanding of biofilm has radically changed our perspective on microorganisms, how they function, and their effect on wound infection.[4]

Practice Point

Antibiotic treatment is based on knowledge of planktonic organisms.

Planktonic organisms of all types float freely in the air and exist on the skin and surfaces in the environment. With injury, organisms contaminate the wound surface. The organisms attach to the surface, change their genetic code (become biofilm genotype), and exude the extracellular polymeric substance (EPS) that provides the structure for the biofilm. Biofilm is defined as communities of microorganisms attached to a surface (living or not) embedded within a hydrated matrix of EPS.[5,6] It is composed of polysaccharide-based substance, although it includes proteins, lipids, and DNA. Biofilm provides protection against antimicrobials and host defenses. The communities of organisms that form are genetically different from each other as well as their planktonic predecessors.[7] The organisms, in the biofilm community, work synergistically, communicate with each other and other communities (through a process called quorum sensing), are able to change their metabolism, and become highly tolerant to antibiotics.[3,8]

The terms “tolerance” and “resistance” have important different meanings to microbiologists. Bacteria that have developed “resistance”

to an antibiotic have a permanent, genetic alteration that enables the planktonic or biofilm phenotypes of the bacteria to survive when exposed to concentrations of the antibiotic that would kill the nonresistant forms of the bacteria. A bacterium that is genetically resistant to an antibiotic is able to survive exposure to the antibiotic regardless of whether the bacteria are growing as single rapidly proliferating planktonic bacteria or if the resistant bacterium is part of a large biofilm community. In marked contrast, the term “tolerance” is used to describe bacteria that have developed the ability to survive exposure to an antibiotic but only when the bacteria are in the biofilm phenotype. When the “tolerant” bacteria in the biofilm community are dispersed back into the planktonic single bacteria phenotype, they become sensitive to the antibiotic again. Thus, the tolerant state of the bacteria to an antibiotic is not permanent and is totally dependent on the unique conditions that exist only in the biofilm community. These unique conditions include the reduced diffusion of antibiotic molecules into all areas of the biofilm structure, and probably most importantly, the frequent reduction in metabolic activity that many bacteria undergo when in a mature biofilm community. This reduced “metabolic state” of some of the biofilm bacteria enables the “metabolically dormant” biofilm bacteria to survive exposure to antibiotics that would rapidly kill the metabolically active planktonic bacteria. This is because, in general, all antibiotics kill bacteria by interfering with bacterial enzyme and protein systems that are essential for bacterial growth, such as energy production (ATP and electron transport), membrane synthesis, membrane transport proteins, or RNA, DNA, or protein synthesis. Thus, if a bacterium is metabolically dormant and not actively using these systems that are essential survival and proliferation, then antibiotics do not have the opportunity to block them and kill the bacterium.[9,10]

Biofilm communities are usually composed of multiple organisms. They form fluid-filled channels that allow oxygen and nutrients to get to the cells and carry away waste. Their ability to reduce their metabolism allows these organisms to use little oxygen and nutrients and become dormant, only later to be reactivated. Biofilms impair healing as the EPS helps the organisms evade the immune system and contribute to perpetuating the inflammatory phase so the wound turns into a chronic nonhealing wound.[5,8]

At some point, the biofilm communities come to exceed their nutrient/oxygen capacity and send off organisms into the wound environment (seed the environment).[11] This process of "shedding" planktonic bacteria or small microcolonies of biofilm containing a relatively small number of bacteria can help to spread the planktonic and biofilm bacteria through wound fluid to new areas of a wound bed and go on to repeat the process of setting up a new biofilm. The newly released planktonic organisms also stimulate a continuation of the inflammatory response. In addition, several major components of the exopolymeric substances (EPS) that constitute most bacterial biofilm matrices are inherently inflammatory. For example, extracellular bacterial DNA (ebDNA) can constitute up to about 20% of the total mass of a mature biofilm EPS. Importantly, toll-like receptor 9 (TLR-9) of the innate immune system of humans has evolved the ability to recognize and bind to unique nucleotide sequences called CpG islands that are present only in bacteria, which leads to rapid recruitment and activation of neutrophils and areas in wounds that have developed biofilms.[12] Similarly, TLS2 and TLR4 that are expressed by epithelial cells of the upper respiratory track can bind the peptidoglycan and lipoteichoic acid from gram-positive and gram-negative bacteria and activate the NF-κβ pathway triggering expression of downstream cytokines, chemokines, and destructive enzymes.[13]

Practice Point

Scientific discoveries have radically changed the way we think of infection. Evidence-based practice requires current scientific knowledge about biofilm as the basis for safe care.

The biofilm generally is not visible because of its location, except perhaps in wounds. Some authors suggest that biofilm in wounds may be observed by expert clinicians as translucent films or opaque yellow-green material.[6] Others do not believe it is visible.[3,5] For the most part,

clinicians can only infer the presence of biofilm genotype organisms based on signs and symptoms. Sophisticated diagnostic tools needed to identify biofilms are not currently available in clinical practice.[3,5]

Seminal work showed that biofilms occur more frequently in chronic wounds than acute wounds (60% of chronic wounds and 6% of acute wounds).[14] Thus, in this chapter where appropriate, the differentiation between acute and chronic wounds is presented.

Practice Point

Biofilm occurs much more frequently in chronic wounds (every 8 out of 10 wounds) than in acute wounds (1 in 12 wounds). The physical characteristics of biofilm have not been identified; the presence of biofilm is inferred from signs and symptoms.

Wound Bioburden Cycle

Predictable processes occur that help us understand how exposure to bioburden can lead to infection or eradication of invading organisms in wounds. Risk of wound infection involves the interaction of the individual's immune system, the environment, and the organisms.[3,15] Increased risk of infection occurs in patients who are immunocompromised as occurs with therapeutic chemotherapy for cancer and persons receiving prednisone. Infection risk also is increased when the environment is moist and warm and lacks appropriate pH and sufficient oxygen and when there is a large load of bioburden present. The nature of the organisms also affects the risk of infection and depends on their constitutional needs such as oxygen requirement (aerobic/anaerobic) and inherent virulence, for example, high virulence as seen in beta-hemolytic *Streptococcus*.

The wound bioburden cycle describes the processes associated with the increasing invasion of a wound by organisms (Table 7-1). The first phase of this cycle is contamination, and it begins with injury that disrupts the skin. Examples of tissue injury are a surgical incision, trauma, or a bite. Microorganisms present in the environment contaminate the wound. The full range of microorganisms may be included (bacteria, fungi, viruses), and they may be endogenous (i.e., from the patient's skin, gastrointestinal tract, or mucous membranes) or exogenous (i.e., from external contamination, e.g., healthcare workers, trauma). Contamination cannot be seen, nor does it generate clinical signs and symptoms. The organisms are planktonic genotype (free floating) and are normally managed by host defenses, and the bioburden cycle terminated at this phase.[3]

Table 7-1 Phases of the Wound Bioburden Cycle

Phase	Definition	Signs and Symptoms
Contamination	Organisms present on wound surface.	No signs or symptoms
Colonization	Organisms multiply on wound surface. Early reversible adherence, later irreversible adherence of organisms to the tissue.	No signs or symptoms
Critical colonization/ biofilm	Organisms form biofilm on wound surface.	Delayed healing. Local infection
Infection	Organisms invade tissue and systemic response occurs.	Fever, leukocytosis, inflammation (redness, warmth, increased exudate, pain, fever, leukocytosis)

Practice Point

Contamination and colonization cannot be seen and produce no signs or symptoms. This phase of the wound bioburden cycle offers the best opportunity for removal of organisms from the wound, before the organisms become attached to the wound surface.

If the microorganisms are not removed, they assess the environment with receptors on their cell wall and attach to the surface. At this point, the organisms' attachment is reversible, that is, the organism can be washed away with irrigation.[3] Once lightly attached, the organisms use the wound bed as a source of nutrition, oxygen, etc. The organisms soon become irreversibly attached, and early stages of biofilm are formed. Over time, the biofilm genotype develops, and the organisms begin to modify their genetic profile and multiply. Colonization cannot be seen and does not produce signs and symptoms.[16]

Within 24 hours of contamination, the organisms begin to develop a thin layer of EPS, and the layer becomes thicker as the biofilm organisms continue to lay down layers of ESP.[6] Biofilm forms during the critical colonization/biofilm phase, and this phase is characterized by local infection and delayed healing.[5,15] In fact, it has been proposed that the concept of critical colonization represents the effects of the

biofilm, so in this chapter, critical colonization is termed critical colonization/biofilm.

Practice Point

The critical colonization/biofilm phase occurs when organisms create local communities in the biofilm on the wound. Local infection and delayed healing are visible to the clinician and characterize this phase.

Infection occurs when the organisms invade the tissue and produce systemic signs. Infection is characterized by inflammation (redness, heat, edema, pain, and increased exudate).[17,18] It is treated with antibiotics, but they are effective only when the organisms are metabolically active.

Practice Point

Infection occurs when organisms invade the tissue and produce systemic signs. Antibiotics used to treat wound infection function only when organisms are metabolically active.

Table 7-1 summarizes the phases of the wound bioburden cycle and their associated signs and symptoms. It should be noted that with the increasing availability of DNA-based diagnostic protocols, a change in this paradigm may be needed that alters the phases of the cycle. One such proposed paradigm is contamination, attachment, proliferation,

critical concentration of signaling molecules, adaptation of the biofilm phenotype, and polymicrobial biofilm.[19]

Defenses Against Infection

Injury mobilizes a predictable, albeit complex, sequence of events that are designed to lead to healing (see Chapter 5, Acute and Chronic Wound Healing). These processes also provide a network of protection against wound invasion by microorganisms. With initiation of the inflammatory response to tissue injury, there is an influx of polymorphonucleocytes (PMNs) and monocytes. Of the PMNs, neutrophils are the most common and arrive immediately after injury and predominate for the first 48 hours.[20] The monocytes enter the wound space immediate after the PMNs and are transformed into macrophages, reaching their peak within 24 hours of injury. Both the leukocytes and macrophages are important in quickly infiltrating the area of tissue damage and removing microorganisms by phagocytosis. These WBCs work best in a well-perfused oxygen-rich environment as phagocytes require oxygen (oxygen burst).[21]

Proinflammatory mediators, reactive oxygen species, proteases, and antimicrobial peptides are released into the wound and result in microbial killing as well as tissue damage. Normally, the processes stimulated by the proinflammatory mediators that lead to inflammation and immune cell activation are reversed when the neutrophils die (apoptosis) and are engulfed by macrophages.[20] In acute wounds, this occurs by about day 5, and wound infection is circumvented.

Practice Point

Inflammation is important in fighting infection early after injury. Continued inflammation over time characterizes chronic nonhealing wounds.

What was a positive response to injury becomes a negative one when the inflammatory cycle continues beyond its normal duration.

Specifically, chronic wounds inflammatory cells continue to be recruited; generate reactive oxygen species, proteases, and antimicrobial peptides that kill microorganisms; and result in tissue damage. The organisms themselves may participate in protective processes, for example, *Pseudomonas aeruginosa* may contribute to inflammation by producing rhamnolipids that protect them from phagocytosis.[22]

Proteases are especially problematic as they cause degradation of the extracellular matrix and tissue damage. A negative spiral is set up where damage caused by neutrophil generated proteases and chronic inflammation delays wound healing (delayed reepithelialization, impaired collagen generation, cells at the edge of the wound).[20,23] The biofilm protective mechanisms are in place, and wound infection occurs when the microorganisms overtake the individual's immune system.

From a clinical perspective, it is important to recognize that the body's defense against microorganisms works effectively in most acute wounds as seen by the low rate (<2%) of surgical site infection (SSI), recognizing that even at this rate, SSI is the most common hospital-acquired complication.[24] Comparatively, chronic wounds present a grim picture with the prevalence of wound infection estimated to be 53%.[25] With more than 40 million inpatient surgeries each year and the increasing incidence of chronic diseases that often involve chronic wounds, for example, diabetes and venous disease, it would behoove us to be vigilant in applying evidence-based care to mitigate wound infection and its sequelae. While the etiology of various types of chronic wounds differ (e.g., diabetic ulcers are due to arteriosclerotic peripheral vascular disease often complicated with peripheral neuropathy; pressure injuries [PIs] are caused by unrelieved pressure), there is agreement in the field that the pathway for chronic wound infection is applicable across the chronic wound types.[3]

Defining Infection

Wound infection is the invasion and multiplication of microorganisms in wound tissue resulting in pathophysiologic effects or tissue injury[26] or the presence on any beta-hemolytic *Streptococcus*.[27] The definition sets out the criteria for a wound infection: tissue must be viable, organisms invade tissue, organisms multiply, patient defenses are overwhelmed, and signs and symptoms of infection are visible. Also, beta-hemolytic *Streptococcus* needs to be identified early because of their well-recognized virulence. Necrotic tissue and slough by definition are dead, so infection cannot be present in them; however, dead tissue does support proliferation of organisms and so needs to be removed.[16,23] Also, pus is the product of phagocytosis and digestion of dead microorganisms by WBCs; pus is not indicative of infection.

Practice Point

Infection occurs in living tissue and so cannot occur in debris; however, debris (slough, necrotic tissue, old sutures) does support proliferation of organisms and so needs to be removed.

The definition of infection was established based on acute wounds and the existing understanding of planktonic organisms. Since then, our understanding of biofilm in chronic wounds and biofilm genotype organism behavior has grown. In addition, the National Institutes of Health (NIH) has declared that 80% of infections are due to biofilm.[28] As invasion during critical colonization/biofilm results in delayed healing, the question must be posed whether wounds in the critical colonization/biofilm phase of the wound bioburden cycle are infected and should be treated as infected. The expectation is that in the near future, the availability of bedside molecular diagnostic techniques will

allow better definition of bioburden and biofilm and development of more effective treatment of offending organisms.[19]

Wound infection currently is diagnosed based on its signs and symptoms.[5,18] Classic characteristics of wound infection are erythema, heat, swelling, and pain.[3,17] One of the challenges in clinical practice is differentiating infection from inflammation. Inflammation normally is present for 3 to 5 days after injury and is characterized by the same signs.[5,18] The question becomes how to differentiate these two processes.

The most common acute wounds are surgical, and the criteria for SSI are used for acute wounds. The definition for SSI was established several decades ago and continues to be the criteria used by the Centers for Disease Control and Prevention (CDC).[29]

Chronic wounds, on the other hand, are characterized by a chronic inflammation and biofilm. However, the identification of infection in chronic wounds is tricky as the signs and symptoms may be more subtle than in acute wounds and indicators of moving from critical colonization to infection may not be recognized.

To date, there are not universal criteria for infection in chronic wounds. Lack of criteria contributes to inconsistent diagnosis of chronic wound infection and potentially to inappropriate treatment (under- or overtreatment). The problem of lack of widely accepted criteria is compounded by the lack of clinical diagnostic tools to evaluate the contribution of the biofilm to infection in chronic wounds.[4,5,30]

Signs and Symptoms of Wound Infection

SSI is divided into incisional and organ/space. Incisional SSI is further divided into superficial and deep SSI.[31] Superficial SSI sites include the skin and subcutaneous tissue while deep SSI designates infection in soft tissues, fascia, and muscle layers of the incision. Organ/space SSIs involve any part of the anatomy other than the incision or that manipulated during the operation (Table 7-2).

Table 7-2 Diagnostic Criteria for Incisional and Organ/Space Surgical Site Infection[31]

	Incisional		
	Superficial[a]	**Deep**	**Organ/Space**
Purulent drainage	From superficial incision	From deep incision but not organ/space	From a drain placed through stab wound into organ/space
Culture	Positive from aseptically obtained fluid or incision from superficial incision		Positive from aseptically obtained culture of fluid or tissue in organ/space
Signs and symptoms	AT LEAST ONE: pain or tenderness, localized swelling, redness, heat AND incision is deliberately opened UNLESS incision culture is negative		
Dehiscence		Spontaneously or is deliberately opened when patient has AT LEAST one: fever (>38°C), localized pain, or tenderness, UNLESS culture is negative	
Abscess		Found on direct examination, reoperation or histopathology or radiology	Found on direct examination, reoperation or histopathology or radiology
MD or surgeon diagnosis	Of superficial incisional SSI	Of deep incisional SSI	Of organ/space SSI

[a]Not superficial SSI: stitch abscess, episiotomy or neonate circumcision, infected burn, incisional SSI that extends into fascia and muscle.

SSI refers to infection at the site of an incision. All SSI occurs within 30 days of surgery, except deep SSI that involves an implant. The time frame for these may extend up to 1 year if an implant is in place, the SSI appears related to the implant surgery, and the infection meets the anatomic criteria for deep SSI.

Practice Point

Diagnosis of wound infection in acute wounds is based on the depth of the infection. In addition, the criteria for infection in acute and chronic wounds are not the same.

While much work has been done by pioneering investigators to identify infection in chronic wounds, most was done before there was an appreciation of the presence of biofilm and its effect on the total wound bioburden. Often cited for their groundbreaking work in diagnosing infection in chronic wounds, Cutting and Harding[17] identified traditional infection criteria (abscess, cellulitis, discharge [serous exudate with inflammation, seropurulent, hemopurulent, pus]), as well as the additional criteria for granulating tissues: delayed healing, discoloration, friable granulation tissue that bleeds easily, unexpected pain/tenderness, pocketing at the base of the wound, bridging of epithelium or soft tissue, abnormal smell, and wound breakdown.[16] The validity of these criteria was tested on ward nurses (n = 20) and compared with wound infection diagnosis by the principal investigator (an MD) and swab culture results. The nurses had less than chance agreement with the MD (47.5%), and the MD had nearly perfect agreement (97.5%) with culture findings.[32] The relationship of the nurses' infection classification with the culture data was not reported. These data indicate that the assessment of the presence of wound infection is not consistent among healthcare providers. The authors concluded that the nurses needed more education to improve their performance.

Practice Point

Practitioners responsible for identifying wound infection need knowledge about the criteria and practice in applying them.

Subsequently, a landmark study by Gardner et al.[18] tested the Cutting and Harding findings.[17] They studied signs and symptoms of infection in patients with chronic wounds (*n* = 36), 11 of whom were infected based on biopsy with both aerobic and anaerobic analyses. Classic signs of infection (pain, erythema, edema, heat, purulence) were compared with signs of infection in secondary wounds (serous drainage with inflammation, delayed healing, discolored granulation tissue, friable granulation tissue, pocketing at the base of the wound, foul odor, and wound breakdown). The Clinical Signs and Symptoms Checklist,[18] comprising the signs and symptoms with operational definitions and mutually exclusive evaluative categories, was used to measure the signs and symptoms (Box 7-1). Findings showed that the signs and symptoms of chronic wounds were better indicators of infection in chronic wounds than were the classic signs (sensitivity 0.62, specificity 0.38). Indicators of infection in chronic wounds were increasing pain, friable granulation tissue, foul odor, and wound breakdown. Specificity of increasing pain (1.0) and wound breakdown (1.0) were robust enough to be considered signs of infection.

Subsequent work by Gardner et al.[33] established the interrater reliability of the Clinical Signs and Symptoms Checklist in diabetic foot ulcers (DFUs) (*n* = 64). Total percent agreement was 76% to 100% (kappa 0.34 to 1.0), establishing the reliability of the scale.

Box 7-1 Clinical Signs and Symptoms Checklist[34*a*]

Clinical Signs and Symptoms Checklist

Signs and Symptoms	Check (+) If Present
Increasing pain in the ulcer area The patient reports increased level of periulcer pain since the ulcer developed. Ask the patient to select the most appropriate statement for current level of ulcer pain from the following choices: **1.** I can't detect pain in ulcer area. **2.** I have less ulcer pain now than I had in the past. **3.** The intensity of the ulcer pain has remained the same since the ulcer developed. **4.** I have more ulcer pain now than I had in the past. If the patient selects number 4, the patient's pain is increasing. Write n/a if the patient can't respond to the question.	☐
Erythema The presence of bright or dark red skin or darkening of normal ethnic skin color immediately adjacent to the ulcer opening indicates erythema.	☐
Edema The presence of shiny, taut skin or pitting impressions in the skin adjacent to the ulcer but within 4 cm from the ulcer margin indicates edema. Assess pitting edema by firmly pressing the skin within 4 cm of ulcer margin with a finger, releasing, and waiting 5 seconds to observe indentation.	☐
Heat A detectable increase in temperature of the skin adjacent to the ulcer but within 4 cm of the ulcer margin as compared with the skin 10 cm proximal to the wound indicates heat. Assess differences in skin temperature using the back of your hand or your wrist.	☐
Purulent exudate Tan, creamy, yellow, or green thick fluid that's present on a dry gauze dressing removed from the ulcer 1 hour after the wound was cleaned and dressed indicates purulent exudate.	☐
Sanguineous exudate Bloody fluid that's present on a dry gauze dressing removed from the ulcer 1 hour after the wound was cleaned and dressed indicates sanguineous exudate.	☐
Serous exudate Thin, watery fluid that's present on a dry gauze dressing removed from the ulcer 1 hour after the wound was cleaned and dressed indicates serous exudate.	☐
Delayed healing of the ulcer The patient reporting no change, or an increase in the volume or surface area of the ulcer, over the preceding 4 weeks indicates delayed healing. Ask the patient if the ulcer has filled with tissue or is smaller around than it was 4 weeks ago.	☐
Discoloration of granulation tissue Granulation tissue that is pale, dusky, or dull in color compared with surrounding, healthy tissue. Note variations of normal, beefy red appearance of granulation tissue.	☐
Friable granulation tissue	

Clinical Signs and Symptoms Checklist

Signs and Symptoms	Check (+) If Present
Bleeding of granulation tissue when gently manipulated with a sterile cotton-tipped applicator indicates friable tissue.	☐
Pocketing at base of wound The presence of smooth, nongranulating pockets of ulcer tissue surrounded by beefy red granulation tissue indicates pocketing.	
Foul odor The ulcer may have a putrid or distinctively unpleasant smell.	☐
Wound breakdown Small open areas in newly formed epithelial tissue not caused by reinjury or trauma indicate wound breakdown.	☐

[a]Adapted with permission from HMP communications.

[a]Adapted with permission from HMP communications.

Practice Point

Signs of wound infection in chronic wounds are increasing pain, friable granulation tissue, foul odor, and wound breakdown; the strongest of these indicators is increasing pain and wound breakdown.

The Gardner[34] research team then went on to examine individually the same signs and symptoms in chronic DFUs, those of the Guidelines of the Infection Disease Society of America (IDSA),[35] and a composite predictor based on all the signs and symptoms of infection.[34] Of the 64 participants, 25 had a DFU with a high microbial load (>10^6). Using the same methods as in the 2001 study, researchers evaluated whether the IDSA predicted ulcers. Findings showed no individual sign was a significant predictor of high microbial load nor was the IDSA

combination. The composite predictor performed moderately well (concordance = 0.645 with 95% confidence interval 0.559 to 0.732).

At the same time, Sibbald and colleagues[15] explored ways to translate existing research findings, so they could be used in clinical practice. They proposed diagnostic criteria using the acronym NERDS for critical colonization (Fig. 7-1) and STONEES for infection (Fig. 7-2). With NERDS acronym, critical colonization/biofilm is present in chronic wounds when three or more of the signs/symptoms are present. With the STONEES criteria, infection was present when any three of the STONEES criteria are observed.

Letter	Key information to know	
NONHEALING WOUND 	• The wound is nonhealing despite appropriate interventions (healable wound with the cause treated and patient-centered concerns addressed). • Bacterial damage has caused an increased metabolic load in the chronic wound, creating a proinflammatory wound environment that delays healing.	• To determine a healing trajectory, the wound size should decrease 20% to 40% after 4 weeks of appropriate treatment to heal by week 12. • If the wound does not respond to topical antimicrobial therapy, consider a biopsy after 4 to 12 weeks to rule out an unsuspected diagnosis, such as vasculitis, pyoderma gangrenosum, or malignancy
EXUDATIVE WOUND 	• An increase in wound exudates can be indicative of bacterial imbalance and leads to periwound maceration. • Exudate is often clear before it becomes purulent or sanguineous.	• Increased exudates needs to trigger the clinician to assess for subtle sign of infections. • Protect periwound area using the LOWE© memory jogger (Liquid film-forming acrylate; Ointments; Windowed dressings; External collection devices) for skin barrier to wound margins.
RED AND BLEEDING WOUND 	• When the wound bed tissue is bright red with exuberant granulation tissues and bleeds easily, bacterial imbalance can be suspected.	• Granulation tissue should be pink and firm. The exuberant granulation tissue that is loose and bleeds easily reflects bacterial damage to the forming collagen matrix and an increased vasculature of the tissue.
DEBRIS IN THE WOUND 	• Necrotic tissue and debris in the wound is a food source for bacteria and can encourage a bacterial imbalance.	• Necrotic tissue in the wound bed will require debridement in the presence of adequate circulation. • Debridement choice needs to be determined based on wound type, clinician skill, and resources.
SMELL FROM THE WOUND 	• Smell from bacterial by-products caused by tissue necrosis associated with the inflammatory response is indicative of wound-related bacterial damage. *Pseudomonas* has a characteristic sweet smell/ green color; anaerobes have a putrid odor due to the breakdown of tissue.	• Clinicians need to differentiate the smell of bacterial damage from the odor associated with the interaction of exudates with different dressing materials, particularly some hydrocolloids. Odor may come from superficial or deep tissue damage, and this should not be relied on along with exudates alone as the only signs of increased superficial bacterial burden.

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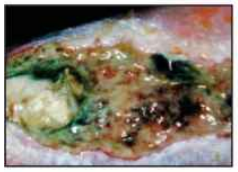
Figure 7-1. NERDS©: Superficial increased bacterial burden.

Letter	Key information to know
SIZE IS BIGGER 	• Size as measured by the longest length and widest width at right angles to the longest length. Only very deep wounds need to have depth measured with a probe. • An increased size may be due to deeper and surrounding tissue damage by bacteria or alternately because the cause has not been treated or there is a systemic or local host factor impairing healing.
TEMPERATURE INCREASED 	• With surrounding tissue infection, temperature is increased. This may be performed crudely by touch with a gloved hand or by using an infrared thermometer or scanning device. There should be a high index of suspicion for infection if >3°F difference in temperature exists between two mirror-image sites.
OS (PROBES TO OR EXPOSED BONE) 	• There is a high incidence of osteomyelitis if bone is exposed or if the clinician can probe to the bone in a person with a neurotrophic foot ulcer. • An MRI is probably the most discriminating diagnostic test and considered necessary for diagnostic dilemmas.
NEW AREAS OF BREAKDOWN 	• Note the satellite areas of skin breakdown that are separated from the main ulcer. • It is important to remember this may be due to the cause of the wound, infection, or local damage being left uncorrected.
EXUDATE 	• All of the features here are due to the inflammatory response. With increased bacterial burden, exudates often increase in quantity and transform a clear or serous texture to frank purulence and may have a hemorrhagic component.
ERYTHEMA, EDEMA 	• The inflammation leads to vasodilatation (erythema), and the leakage of fluid into the tissue will result in edema.
SMELL 	• Bacteria that invade tissue have a "foul" odor. There is an unpleasant sweet odor from *Pseudomonas* gram-negative organisms, and anaerobe organisms can cause a putrid smell from the associated tissue damage.

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Comments

- Clinicians need to have a consistent approach to measurement.
- An increased size from bacterial damage is due to the bacteria spreading from the surface to the surrounding skin and the deeper compartment. This indicates that the combination of bacterial number and virulence has overwhelmed the host resistance.

It is important to distinguish between infection and the other two potential causes of temperature change:

- A difference in vascular skin supply (decreased circulation; is colder).
- Inflammatory conditions are not usually as warm, but they can demonstrate a marked increase temperature with extensive deep tissue destruction (acute Charcot joint).

- Radiographs and bone scans are less reliable for diagnosis of osteomyelitis with loss of bone mass that occurs with neuropathy. Radiographs of well-calcified bone, such as pressure ulcers of the pelvis, may be more reliable. The majority of ulcers that probe to bone in other locations are less likely to be associated with osteomyelitis.

- Search for the cause of the satellite areas of breakdown and the need to correct the cause.
- Check for local damage and consider infection, increased exudates, or other sources of trauma.

- For exudates control, determine the cause and then match the absorbency of the dressing (non, low, moderate, heavy) to the amount of exudates from the wound.
- Assess surrounding skin to evaluate for maceration. Again, use the LOWE© memory jogger (Liquid film-forming acrylate; Ointments; Windowed dressings; External collection devices) for skin barrier for wound margins.

- For erythema and edema control, the cause or the tissue infection needs to be treated.

- Make sure the smell is from organisms and not from the normal distinct odor from the interaction of exudates with some of the dressing material.
- Systemic antimicrobial agents are indicated that will treat the causative organisms, and devitalized tissue should be aggressively debrided in wounds with the ability to heal.

Figure 7-2. STONEES©: Deep compartment infection.

A cross-sectional validation study of patients with leg and foot ulcers conducted by Woo and colleagues[36] found that the presence of any three NERDS signs had a sensitivity of 73% and a specificity of 80.5% when compared with semiquantitative swab cultures with scan or light growth. They also found that any three STONEES signs had a sensitivity of 90% and a specificity of 69.4% when compared with semiquantitative swab cultures with moderate-to-heavy growth. It should be noted that the limitation of this study is that the comparison is with a surface measure of bioburden, rather than a measure of bioburden in the tissue. Also, as noted before, standard clinical microbiology laboratories typically only measure planktonic bacteria and do not measure biofilm bacteria. In addition, a substantial amount of biofilm bacteria are not located on the surface of chronic wounds beds, but actually reside from 30 microns to 60 microns beneath the surface of chronic wound beds and are not accessible by standard wound swabs.[37]

Practice Point

Any three NERDS signs are indicative of critical colonization/biofilm, and any three STONEES signs are indicative of wound infection.

Another early research team, that of Cutting and colleagues,[38] sought expert opinion from international interdisciplinary wound experts to determine the clinical signs and symptoms of infection present in chronic wounds as well as those specific to a particular type of wound. Data showed that signs and symptoms common to all chronic wounds (not acute wounds or burns) were cellulitis, malodor, pain, delayed healing, wound deterioration or breakdown, and an increased amount of exudate. Signs or symptom-specific criteria for infection in various

chronic wounds are crepitus for pressure ulcers; phlegmon (acute inflammation of soft tissues) for neuropathic/diabetic ulcers; increase in local skin temperature for venous ulcers; and dry necrosis (that may become moist and boggy at the edges of the necrotic tissue) for arterial disease–associated tissue breakdown.

Practice Point

Expert opinion of international wound experts using the Delphi technique showed that signs and symptoms of infection in chronic wounds were cellulitis, malodor, pain, delayed healing, wound deterioration or breakdown, and an increased exudate.

A review by Reddy et al.[25] summarizes the literature that examines the relationship between deep tissue biopsy (gold standard) and signs and symptoms of wound infection in patients with chronic wounds. Likelihood ratios (LRs) were used to identify the presence of infection. A positive LR predicts the presence of infection while negative values indicate that wound infection is not present. The likelihood of infection increases when an ulcer results in increasing pain (LR range 11 to 20); however, absence of pain is not as helpful in identifying infection (LR 0.64 to 0.88).[18,34] Classic signs of wound infection were not helpful in diagnosing chronic wound infection (LR small and including one).[18,34]

Practice Point

Classic signs and symptoms of wound infection cannot be used to identify infection in chronic wounds. The presence of pain was helpful in identifying infection in chronic wounds.

When Reddy et al.[25] examined the literature, they found few studies that systematically examined signs and symptoms and compared them with tissue biopsy culture. Lack of using the gold standard was a major issue. For example, the often-cited work of Cutting and Harding[17] proposed signs and symptoms of wound infection based on their clinical experience. In their subsequent work that examined the ability of nurses to identify wound infection based on their list of signs and symptoms, the standard of comparison was MD evaluation; swab cultures were used to test MD diagnosis based on the signs and symptoms from the list he had generated.[32] In the Delphi study that assessed the opinion of experts on signs and symptoms of chronic wound infection,[38] the signs and symptoms elicited were not tested against a standard criterion.

Reddy's team noted that the majority of studies had examined a single sign or symptom and its relationship with criteria whose validity had not been established. They also acknowledged that many of the signs are quite subjective (e.g., friable granulation tissue; odor) and interrater reliability had not been reported. Reddy et al.[25] concluded that data from the Gardner research team provides the strongest evidence for the relationship between signs and symptoms and tissue biopsy cultures. Data from the Sibbald[15] paradigm and the validation study of Woo and colleagues[28] were not considered in the review because they did not compare the signs and symptoms with tissue biopsy.

It is important to note that biofilm is not explicitly identified as a sign of infection in chronic wounds. At this time, there are no routine diagnostic tests available to identify biofilm.[9,10,30] Concern has been raised that biofilm often may be confused with slough, debris, and exudate when it merely provides a surface for biofilm to grow on rather than itself being biofilm.

An international biofilm consensus group[9] reported strong agreement that the clinical signs and symptoms of biofilm were failure of the wound to respond to systemic antibiotics or local antiseptics when guided by antibiotics or antiseptics whose choice was driven by culture. Other signs noted were intermittent flares of inflammation or

infection; high levels of exudate due to inflammation; and gelatinous material on the wound edge that quickly reforms after removal. There was weak agreement that delay in healing was a sign of the presence of biofilm.

Biofilm presence is inferred from treatment failure when using antibiotics and antiseptics prescribed based on culture. Slough, debris, and exudate seen in chronic wounds are not biofilm.

Practice Point

Biofilm is considered present when a wound fails treatment with culture-driven antibiotic/antiseptic treatment.

Diagnostic Tests

Wound Cultures

Tissue biopsy is the gold standard for diagnosing wound infection. Those with greater than 10^5 CFU have been recognized as diagnostic of wound infection.[39] Krizek et al.[40] showed that skin grafts did not "take" when there were more than 10^5 organisms per gram of tissue. Similarly, Robson et al.[41] found that bacteria that exceeded 10^5 per gram of tissue inhibited successful secondary closure of wounds. Classic work of Robson and Heggers[39] using a sample of wounds (*n* = 41) of mixed etiology showed that the quantitative tissue biopsy method had a sensitivity of 100%, a specificity of 93.5%, and an accuracy of 95.1% in predicting the success of delayed closure, laying the foundation for biopsy as the gold standard for infection. Subsequent work revealed that the virulence of beta-hemolytic *Streptococcus* (type A and D) at lower levels resulted in surgical infection and so was added to the definition.[27]

Practice Point

Tissue biopsy with $\geq 10^5$ or the presence of beta-hemolytic *Streptococcus* is the criterion for wound infection in acute wounds.

Today we recognize that wound cultures measure planktonic organisms and underestimate the total number and type of organisms present in a given wound.[9] In addition, biofilm is not uniformly distributed across the wound surface nor are organisms or microcultures of organisms uniformly distributed. Because tissue culture is taken at a single location in the wound, tissue culture cannot accurately capture the bioburden in a wound. This is a problem as

there currently are no routine diagnostic tests available in clinical practice to identify the presence of biofilm in a wound.[9,10,30]

Practice Point

There currently are no routine diagnostic tests available to identify biofilm. Wound culture does not accurately identify the presence of biofilm.

Wound Culture Techniques

Three approaches to culturing are available: biopsy, aspiration, and swab. Table 7-3 describes important dimensions of the procedure.

Table 7-3 Comparison of Three Techniques for Wound Culture

	Biopsy	Aspiration	Swab with Levine Technique
Performed by	MD or provider with appropriate authority and preparation in biopsy	MD or provider with special education in technique and understanding of anatomy	Nurse or physical therapist that has been taught how to collect specimen
Location of specimen	Clean granulation tissue in wound bed	Intact skin adjoining wound	Clean granulation tissue in wound bed
Cleansing before sampling	Best taken immediately after debridement; if not, rinse with saline that has no preservative	Intact skin will be inserted and cleansed with antibacterial solution	Rinse with saline that has no preservative
Use of local anesthetic	Contraindicated	Contraindicated	Not needed
Obtain sample	Tissue sample (at least 0.25 g) removed aseptically with scalpel or punch biopsy. No local anesthetic	Using a 10-mL syringe with #22 safety needle, insert into tissue and aspirate, moving the needle tip at 2–3 angles for directions to obtain 0.5 mL of fluid	Rotate sterile culture tip in 1 cm^2 area for 5 s until wound fluid is elicited
Specimen care	Insert into sterile container, insuring appropriate approach for aerobic/ anaerobic analyses.	Remove excess air, remove needle with hemostat, and after disposing of it in sharps container, cap syringe with sterile blood gas cap	Insert culture tip into sterile container, insuring that portion in the container is sterile. Insure media is appropriate for aerobic/ anaerobic analyses
Transport	Transport immediately. Delay in transport may lead to inaccurate data as organisms can grow in the culture container.		

Regardless of the culture technique employed, preparation of the lab slip that accompanies the specimen is critical to accurate interpretation of the culture findings. The lab needs to know the site on the body where the specimen was obtained, the type of wound, the time the specimen was obtained, the age and gender of the patient, whether receiving antibiotics (name, dose, started when), as well as any special information, for example, occupation (farmer vs. ocean diver), anticoagulated. Prompt delivery of the specimen also is essential so organisms do not have time to grow. Failure to complete these seemingly mundane aspects of wound culture can result in false-positive or false-negative results.[42]

Practice Point

Accurate wound culture data depend on aseptic collection of the specimen, essential data about the wound and patient that are incorporated into the lab slip, and timely transport of the specimen to the lab.

Specimens can be processed qualitatively, quantitatively, or semiquantitatively. Qualitative processing results in isolation and identification of the organisms present. The quantitative processing allows the specific number of organisms grown in media to be identified and counted. The number of organisms is expressed as number per gram of tissue, milliliter of fluid, or swab. The number of organisms present provides information regarding the rate of microorganism multiplication. Few facilities are equipped to perform quantitative analyses, and most are restricted to research studies. The majority of cultures are processed using semiquantitative technique where the cultures are inoculated onto media using a sterile loop that dilutes the specimen from the first to the fourth quadrant. Results are reported as 1+, 2+, 3+, and 4+ (trace, few, moderate, abundant).[42,43] With semiqualitative analyses, authors[15,45] suggest that 4+ should be used as the cutoff for diagnosing infection. The sensitivity of the organisms isolated to antibiotics can be obtained, regardless of which processing technique is used. Normally, results from a culture are available in 24 to 48 hours. Also, analyses for anaerobic organisms and fungi are normally not performed unless a specimen has been obtained using appropriate techniques and the lab has been requested to perform analysis for anaerobes.

Each of these wound culturing techniques has strengths and limitations. Tissue biopsy is the gold standard and is the standard against which the other approaches are compared. However, it is a labor-intensive process where the tissue must be weighed, homogenized, and serially diluted prior to inoculating media and then

plating it and growing it in aerobic and anaerobic conditions.[42] Biopsy also is limited because it causes patients pain when performed, many facilities do not have the resources to perform the laboratory analysis, and it requires that an MD or specially a provider with appropriate education perform it.[42,43]

Few studies have been performed that address aspiration wound culturing. A study by Lee et al.[44] examined aspiration but did not compare it with biopsy. Another found that aspiration underestimated infection identified by biopsy but that the organisms obtained were not consistent with the organisms identified with deep tissue biopsy.[45] Currently, there are insufficient data about the use of aspiration wound cultures to recommend it for culturing wounds, although it is appropriate for assessing the organisms present in and draining abscesses.

Swab cultures have been extensively studied in an effort to identify a less invasive but accurate method of culturing wounds.[46] Early work by Levine et al.[47] showed a high concordance (R^2 = 0.76) between their specific method of obtaining a swab culture (1 cm^2 area, 5-second culture, obtain bloody fluid) and biopsies of the same sites (Fig. 7-3). Other approaches to surface cultures have been used, most recently the Z-stroke where the tissue is swabbed across the wound from edge to edge in a Z pattern. The Levine technique sensitivity was 90% and specificity 57%, while the Z-stroke technique had a sensitivity of 90% and specificity of 27%. The concordance of the Levine technique with tissue biopsy was identical (78%), and concordance with respect to recovering the specific organisms *Staphylococcus aureus*, *P. aeruginosa*, and beta-hemolytic *Streptococcus* was 96%, 96%, and 99%, respectively.[43]

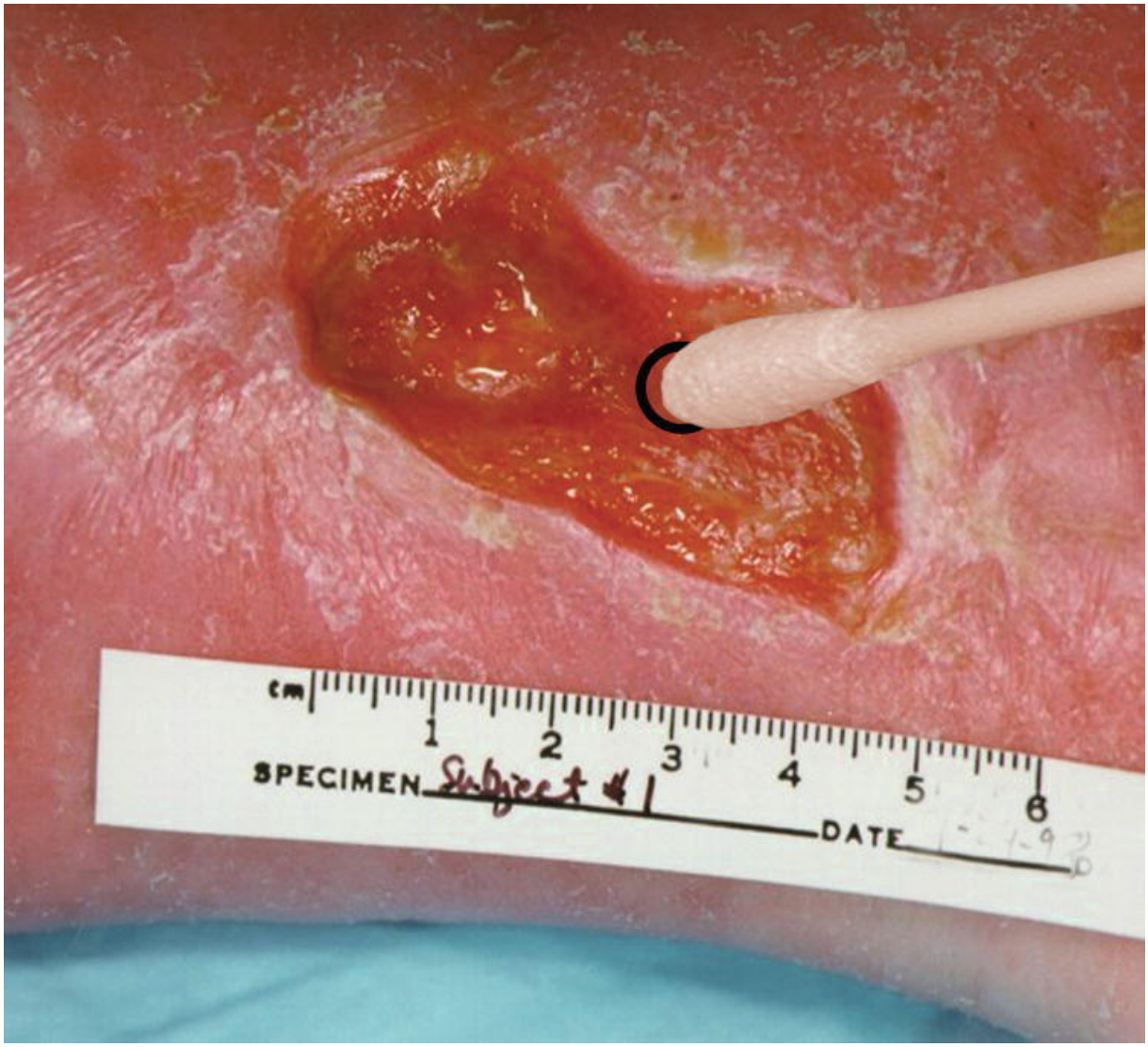

Figure 7-3. The Levine technique. (Photo courtesy of Dr. Rita Frantz.)

Swab culture has the advantages of being easy to perform and requiring minimal training to ensure appropriate sampling. However, it accesses wound surface organisms. It does not sample organisms deep in the tissue, including anaerobic organisms.[9] An additional threat to accuracy is the possibility of dry swabs or swabs with insufficient specimen.[42] One approach proposed to overcome the concern of contaminants is wound cleansing, recommended to remove exudate, topical therapies, and loose nonviable tissue prior to obtaining

the culture. Moistening the swab with normal saline or transport medium is recommended by some prior to specimen collection to address the potential dryness issue.[46,48]

Although the accuracy of swab cultures as compared with biopsy cultures has been studied, methodological issues interfere with our ability to use this information as the basis for clinical practice.[46,47,49] The most serious methodological problem presented by these studies is that the specific swabbing techniques employed were not described. Swabbing techniques vary greatly according to wound preparation, area of the wound sampled, duration of sampling, and even the type of swab employed (e.g., alginate).[50]

To address this problem, Gardner et al.[43] compared culture findings from swab specimens obtained from wound exudate, the Z-stroke technique, and Levine's technique with culture findings from viable wound specimens. Culture findings based on swab specimens obtained using Levine's technique were more accurate and concordant with culture findings based on tissue specimens than swabs taken with either wound exudate or the Z-stroke technique.

At a critical threshold of 37,000 organisms per swab, the Levine technique had a sensitivity of 90% and specificity of 57% when the true infection status of the wound was defined as 1,000,000 or more organisms per gram of tissue. The Z-stroke technique using the critical threshold of 62,500 organisms had a sensitivity of 90% but only a 27% specificity. In terms of recovering any and all organisms, swab specimens obtained using Levine's technique were 78% concordant with tissue specimens.

A systematic review by Copeland-Halperin et al.[51] compared swab and biopsies to culture in infected wounds. Their findings showed that the Levine technique was superior to the Z-stroke technique, and they suggest it may be appropriate for initial wound monitoring. They also found that punch biopsies provide better information (sensitivity 100%, specificity 90%, and accuracy 95%) than do the other techniques for antibiotic-resistant wounds.

Recent work by Haalboom et al.[52] compared culture using the Levine technique with wound biopsy (punch biopsy 3 mm) in chronic wounds (n = 180). Aerobic and anaerobic cultures were obtained.

Identical results were seen in 93 wounds (51.7%) when skin flora was included and in 131 (72.8%) when skin flora was excluded. Highest agreement was for *Escherichia* (99.4%, kappa 0.93) and lowest for *Staphylococcus* sup (84.4%, kappa 0.66). The authors conclude that the techniques yield similar results and recommend that clinicians initially use the Levine technique for culturing because of the more modest patient burden. They suggest that biopsy should be reserved for patients who do not respond to antibiotic treatment based on swab culture and sensitivities. Haalboom et al.[52] go on to remind clinicians that neither technique measures the entire bioburden of the wound.

Evidence-Based Practice

Swab cultures should be obtained using the Levine technique as it obtains fluid from the wound tissue, not just surface organisms.

The Reddy et al.[25] review was conducted in part to determine the preferred noninvasive method of culturing chronic wounds. Few (four) studies met the inclusion criteria that included comparison of the techniques with tissue biopsy, and they calculated LRs to identify predictors of infection (positive LR) and predictors of no infection (negative LR). The Gardner study[43] that examined nonarterial chronic wound ulcers using the Levine technique (when compared with biopsy) had an LR of 6.3 (95% CI 2.5 to 15), showing its strength in identifying infection. Similarly, a negative wound culture (LR 0.47 with 95% CI 0.31 to 0.73) indicated that infection was less likely. In contrast, the Z-stroke technique neither predicted nor excluded wound infection. In a later study by Gardner et al.[34] on patients with nonarterial diabetic ulcers, Levine technique did not predict infection, but negative cultures predicted the absence of infection.

Implications of the work of Copeland-Halperin et al.[51] and Haalboom et al.[52] are that surface cultures may be appropriate for

initial monitoring of wound status. Haalboom suggests that tissue biopsy should be reserved for patients with wounds that do not respond to treatment. While clinical resources and concern about patient burden with biopsy may underlie this recommendation, the most accurate culture needs to be used and that is tissue culture.

In many ways, this review summarizes the existing literature about noninvasive wound culture in patients with chronic wounds. First, the prevalence of chronic wounds is high, especially in spinal cord–injured patients, long-term care residents, and diabetics. Wound infection in the chronic wound populations is high, the range depending to some extent on the quality of the research.[25] Yet, even the high prevalence of infection in patients with chronic wounds may be an underestimation as patients with infected wounds often are excluded from many research studies. This review shows that the identification of wound infection remains challenging. Clinical signs and symptoms of infection are important in wound assessment. Inflammation markers are especially important.

However, the signs and symptoms associated with inflammation may not be present in some patients with acute wounds or in patients with chronic wounds. The signs specific to chronic wounds continue to require ongoing attention. Wound pain and wound breakdown need to be routinely incorporated into assessment. Consideration also needs to be given to routine evaluation of a set of signs and symptoms of infection in chronic wounds such as is provided by the Clinical Symptom Assessment List[18] and the Sibbald et al.[15] NERDS and STONEES approach. The use of criteria that have established operational definitions will aid providers in evaluating chronic wounds consistently.

Wounds suspected of infection, especially those with delayed healing, are often cultured to confirm the diagnosis. While qualitative cultures provide useful information in wounds that are demonstrating obvious clinical signs of infection, they may not be as useful in diagnosing infection in the absence of signs and symptoms unless certain pathogens are isolated. In the absence of clinical signs and symptoms, quantitative cultures remain the gold standard method for diagnosing localized wound infection. Newer techniques on the horizon

hold hope for a revolution in point of care diagnostics for wound infection.

Newer Techniques to Identify Bacteria in Wounds

Advanced techniques have been used in research to evaluate the presence of biofilm and the nature of organisms in it. Wu et al.[53] suggest current diagnostics can be classified as including morphology assays (tissue sampling, confocal microscopy, scanning electron microscopy); microbiological assays (standard clinical culturing methods, and sonificantion or dithiothreitol treatment if a prosthesis is present); and molecular assays (16S RNA qPCR, bacterial tag-encoded FLX amplicon pyrosequencing, PRAPS [partial ribosomal amplification and pyrosequencing], FRACS [full ribosomal amplification, cloning, and Sanger sequencing], PRADS [partial ribosomal amplification, denaturing gradient gel electrophoresis]). Each has assets and limitations. Importantly, 16S rRNA PCR and bacterial tagging with FLX amplicon pyrosequencing allow identification of the pathogen based on genetic analysis but are unable to differentiate planktonic from biofilm bacteria. In addition, all these methods are expensive and most available primarily for research purposes.

Clinical identification of biofilm is challenging. One approach, blotting, has been tested in patients with pressure ulcers.[54] With this approach, a membrane is placed briefly over a newly debrided or cleansed wound bed. The membrane is subsequently stained, and data can predict the development of slough in the week following the blotting. The presence of slough is thought to have come from biofilm. These preliminary data suggest blotting can be used clinically to identify the areas where biofilm still exists. The recognition that most chronic wounds have high levels of proteases has led to the development of point of care detectors for use with wound fluid. While approved for use in some European countries, these sensors are not currently available in the United States.[55]

Pivotal to further work on biofilm is the development of improved diagnostics. Much research is focused on this area, and clinicians will need to continue to update their knowledge to provide evidence-based care.

Practice Point

A bedside test for identifying microorganisms in biofilm does not currently exist. Ongoing research continues to develop and test this needed diagnostic tool.

Treatment of Bioburden, Biofilm, and Infected Wounds

Wound bed preparation has been the cornerstone of managing wounds with delays in healing for nearly two decades.[56,57] Fundamental to the paradigm is a commitment to treat the whole patient. Central to this approach is evaluation and optimization of the patient's basic physiological needs for healing including oxygenation and perfusion. There also is a focus on using the best science to understand and treat the underlying cause(s) of the wound pathology, be it off-loading with DFUs, glucose management, or compression for venous ulcer disease. There is understanding that chronic wounds are distinct from acute wounds in that chronic wounds are characterized by defective extra cellular matrix, prolonged inflammation, and failure to epithelialize. Often a large portion of the problem is bioburden. The goal of care is to return the chronic wounds to an acute wound where growth factors, cytokines, proteases, and their inhibitors are in balance. Therapy is aimed at optimizing tissue, reducing infection/inflammation, balancing wound moisture, and facilitating epithelial edge advancement—the TIME paradigm. This paradigm is described in Chapter 5, Acute and Chronic Wound Healing.

As our understanding of biofilm has increased, there is a need for attention to its role in delayed healing and/or infected wounds. Recently the step-down/step-up model of care for patients who are at risk for or actually have increased levels of biofilm was proposed. It is a bundled approach that is applicable to wounds that are chronic and acute wounds (healing by secondary intention), and those that are infected.[9,10,30]

Such a bundled approach to chronic wound management was tested by Wolcott and Rhoads[58] who evaluated the frequency of complete healing in patients with chronic wounds who had critical limb ischemia. Patients were individually managed to address systematic threats to healing. Wound treatment was guided by the physical appearance of the wound, and care was individualized to include the entire patient. Treatment was focused on specific goals and included blocking biofilm attachment (lactoferrin, EDTA), killing bacteria

(antibiotics, silver, iodine), inhibiting quorum sensing (farnesol), interfering with EPS (farnesol, xylitol), and addressing false metabolites (gallium, xylitol). In this retrospective study, 77% healed, a much higher rate than other studies. The authors conclude that treating biofilm with multiple treatments simultaneously is needed. However, there is a need for prospective research to confirm these findings.

Pivotal to the step-down/step-up model is the early and aggressive treatment of wounds. Debridement, local and systemic antibiotics (empiric), management of the overall condition of the patient and the specific etiology of the wound (host factors), and early identification of the organisms on the surface and biofilm form the foundation for subsequent care. The themes of debridement, optimizing antiseptics/antibiotics, continued assessment of healing/inflammation, and management of host factors continues until the wound is healed or is not progressing as planned and so adjunctive therapies are instituted or modified. Inherent in this model is early implementation of biofilm disrupting technology. This biofilm disruption concept continues in those who have continued impaired healing and may be augmented with more aggressive or different adjunct treatments implemented overtime.[10]

How widely this model it is applied will depend on the clinician who may apply it to all, to those with high risk, or not at all. The ability to use the model will depend to some extent the available resources, for example, molecular analysis.

The combination of wound bed preparation and the step-down/step-up biofilm model provide a contemporary approach to addressing increased levels of bioburden, biofilm, and infection. The step-down/step-up model front-loads treatment and proposes a time frame for action, reflecting recognition that the window of opportunity to keep planktonic organisms from attaching to the wound surface is limited and failure to disrupt that attachment can result in the development of a mature biofilm that is tolerant to antibiotics.

The first step in the step-down/step-up model is the initiation of multiple therapies in combination on days 1 to 4.[10] Therapies addressed include debridement, empiric systemic antibiotic and topical antiseptics, biofilm disrupting technology, management of host factors, and identification of biofilm DNA. The second phase occurs on about

days 5 to 7, and the focus here is on personalizing therapy based on wound status. Inflammation and healing status are evaluated, biofilm disruption technology is continued/changed, topical antiseptics and antibiotics are initiated based on the diagnostic work completed in the first phase, and there is continued management of host factors. The third phase occurs from about weeks 1 to 4 and the focus is on reevaluating and adjusting therapy depending on inflammation and healing status. The same parameters are evaluated in the fourth phase and if healing is taking place, therapies (biofilm disruption technology, antibiotics/antiseptics, etc.) may be “stepped down” and the wound care moved to standard care until healing is complete. If inflammation and healing have not improved or have deteriorated, therapies are “stepped up” and advanced therapies initiated as appropriate.

Debridement

At the stage of critical colonization/biofilm, sharp/aggressive debridement is needed. The goal of debridement is to remove all devitalized tissue as it provides an excellent medium for growth of microorganisms as well as existing biofilm and with biofilm genotype organisms. This is somewhat of a challenge as actual biofilm is not visible to the provider and even if surgical debridement is provided, it does not remove all the biofilm.[10]

Sharp debridement is needed to open tunnels and to address undermined areas, should they exist.[16] With the viable tissue exposed and the biofilm disrupted, there is a window of opportunity within the first 24 hours after debridement where topical agents can be used to kill the planktonic organisms before they become attached to the wound surface. Within 24 hours of debridement, biofilm again begins to develop, resulting in a mature biofilm within 72 hours. Sharp debridement reduces biofilm burden, and its repeated use is recommended at a minimum of every 7 days.[16] While bedside or office debridement can be performed on some patients (those with small wounds, not anticoagulated, no comorbidities, minimal pain), some authors recommend taking the patient to surgery for debridement.[59] Better pain control, management of hemostasis, and more extensive debridement are advantages of taking the patient to surgery for debridement.

An important issue in the recommendation for surgical/sharp is the availability of trained personnel to perform adequate debridement to interrupt the wound–bioburden cycle. The provider who performs the debridement must have the knowledge, skill, and authority to safely perform sharp debridement.[16] Referral of the patient to a wound center or outside consultant may be needed in some situations, for example, provider with limited experience with wounds, lack of resources. Management of patients with chronic wounds is complex, and practitioners with expertise, support staff, and essential equipment are needed to facilitate healing.[59]

Practice Point

Sharp debridement repeated at weekly intervals reduces biofilm bioburden and is first-line treatment in antibiofilm treatment.

A recent systematic review examined hydrosurgery, ultrasound therapy, and plasma-mediated bipolar radio frequency ablation therapy (coblation).[60] The authors concluded that ultrasound reduced exudate and improved wound healing times. For hydrotherapy and coblation, there was a lack of randomized clinical trials (RCTs) comparing these approaches with each other or with standard techniques.

The effectiveness of another new technology, a monofilament debridement pad, was evaluated for the management of wounds with slough[61] Using an ex vivo porcine model, the pad was effective in slough removal. Subsequent preliminary evaluation was conducted in a sample of 10 patients with wounds with slough. After 4 weeks, mean wound size decreased, exudate was reduced, and healing improved. Further work is needed, but a mechanical alternative to surgical debridement is attractive.

Enzymatic debridement has been be used to remove devitalized tissue, but its value on biofilm has yet to be established.[3] In addition, it is recognized as slower but less traumatic to tissues. It is not recommended for biofilm removal.[9]

Some authors recommend the use of biodebridement with maggots as part of the wound bed preparation paradigm.[62] Dumville et al.[63] compared the clinical effectiveness (time to healing, time to debridement, bacterial load resistance of methicillin resistant *S. aureus*, pain) in patients (n = 267) with venous or mixed venous–arterial ulcers. They found that the time to debridement was less and pain was higher in those receiving the maggot therapy versus a hydrogel.

In an RCT, Opletalová et al.[64] compared the efficacy of bagged larvae in wound debridement compared with conventional treatment in nonhealing wounds with slough (n = 119). The larvae were more effective by day 8 (P = 0.04), but by day 15, there was no difference in slough (P = 0.78), suggesting that they are effective at 1 week, but there is no benefit to continue the therapy after 1 week. A larval therapy foam dressing also was compared with a standard larvae removal therapy (hydrogel) in patients with venous ulcers or mixed ulcers.[65] The larva therapy dressing performed better with debridement of 31/32 compared with the hydrogel (11/32) (P = 0.001). The experimental dressing group had fewer dressing changes (P = 0.001) but had greater pain (P = 0.001).

A recent systematic review by Sun and colleagues[66] concluded that the use of maggots increased wound healing and decreased time to healing. Some authors recommend the use of biodebridement with maggots as part of the wound bed preparation paradigm because it aids debridement and the larvae have anti-inflammatory and wound healing properties.[62] Further work will be required before larvae therapy replaces sharp debridement, especially since its full effects are not seen for several days. Wolcott[16] recommends that surgical debridement be combined with topical antiseptics and irrigation to maximally remove biofilm organisms. Details on methods of wound debridement are addressed in Chapter 8.

Cleansing

Wound cleansing and/or irrigation are important after debridement[16] as well as with each dressing change.[3,67] Cleansing of wounds is performed to reduce planktonic bacteria, foreign debris, contaminants, and inflammatory exudates present on the wound surface that may create tissue damage. Wound cleaning is a process that removes these less adherent inflammatory contaminants from the wound surface and renders the wound less conducive to microbial growth. On the other hand, cleansing itself may create tissue damage, depending on the pressure used for cleansing and the cleansing agent. Host factors such as foreign debris and contaminants on the surface of the wound can harbor microorganisms or provide nutrients for their growth.

A Cochrane meta-analysis of patients with either acute or chronic wounds examined the effect of cleansing of the wound surface on infection and healing. They compared normal saline versus tap water and tap water versus no cleansing.[68] Data showed no difference in infection rates with normal saline versus tap water and no difference with cleansing with tap water versus no cleansing. Healing rates also did not differ. The authors noted that the lack of consistent definition for infection was an important limitation in this meta-analysis. They also emphasized that tap water had to be safe when used. Theoretically, water should not be used because of its deleterious effects on open tissue; however, because the fluid is only briefly in contact with the wound surface, it isn't crucial that the solution be isotonic (0.9% sodium chloride). If an isotonic saline solution is used, however, it can be prepared inexpensively by combining 1 teaspoon of noniodized salt in 1 quart of water.

Patient Teaching*Preparing Saline Solution at Home*

To prepare saline solution at home, tell the patient to combine 1 teaspoon of noniodized salt with 1 quart of distilled water, stirring until the salt is completely dissolved. The solution can be stored for up to 1 week, at room temperature, in a tightly covered glass or plastic container.

When open wounds are exudative or full of debris, cleaning with a commercially available wound cleanser should be considered. Many of these products have a surfactant in them to help reduce surface tension and aid in the removal of contaminants that have the potential to cause local tissue damage and inhibit healing. With that said, manufacturers of commercially available wound cleansers are challenged to create a product that has the cytotoxicity level needed to kill microorganisms but without damaging cells needed for normal healing. Studies have examined the toxicity of various wound cleaner and skin cleaners on various cells using a toxicity index, a measure of the product's ability to kill microorganisms without damaging cells needed for repair. To date, an optimal toxicity level has not been proposed. Data show that toxicity is variable by product and type of cell tested (Table 7-4). Also skin cleaners (data not shown) have a higher toxicity index than do wound cleansers[69–71] and are harmful if used to cleanse wounds.

Table 7-4 Comparison of the Toxicity Index of Wound Cleansers in Three Studies

	Foresman et al.[69]	Wilson et al.[70]		Rani et al.[71]
Cells used as basis for toxicity index	WBCs	Fibroblasts	Epidermal keratinocytes	Fibroblasts
Shur-Clens	10	0	0	0
Biolex	100	10	0	10
SAF-Clens	100	0	10	100
Carra-Klenz	100	10	10	10
Dermal wound cleanser	10,000	10	100	10
Puri-Clens	10,000	10	100	X
Restore	X	100	100	0

X, not tested.

Practice Point

Cleansing with each dressing change is recommended. The best agent to use for cleansing has not been established.

Irrigation

Irrigation is utilized as a strategy to remove planktonic phenotype organisms before they become attached to the EPS. Wound irrigation creates hydraulic forces generated by the fluid stream that are effective in cleaning the wound when the force of the irrigation stream is greater than the adhesion forces that hold the organisms and debris to the wound surface. An ideal pressure for wound cleansing is 5 to 15 psi.[72–74]

A pressure less than 5 psi is ineffective in cleansing. Research on experimental wounds in the animal model found that irrigation pressures of 1 and 5 psi removed significantly less wound debris than did pressures of 10 and 15 psi.[75] A bulb syringe (0.05 psi) used to treat trauma wounds within 24 hours of injury was less effective than pressures of 13 psi created with a needle and a syringe.[76] Significantly more inflammation and wound infection was seen in those wounds cleaned with the bulb syringe.

Evidence-Based Practice

Research indicates that the optimum pressure for wound cleansing is between 5 and 15 psi.

The use of a needle and syringe to deliver fluid to wound tissue is generally regarded as a convenient method of providing effective irrigation pressure. A 35-mL syringe and a 19-gauge needle or angiocatheter has been shown to deliver an irrigation stream at 8 psi[77] and was more effective than a bulb syringe in removing wound bacteria and preventing the development of infection in experimental wounds.[76]

A variety of needle and syringe combinations can be used to achieve the desired range of irrigation pressure. The size of the syringe and the needle gauge determine the amount of pressure of the fluid stream. Since the force depressing the plunger is distributed over a larger surface area, the larger the syringe, the less the force. With a 19-gauge needle, 6-, 12-, and 35-mL syringes will produce pressures of 30, 20, and 8 psi, respectively. The opposite effect occurs by increasing the size of the needle. Since the larger the lumen of the needle, the greater will be the flow of fluid, needles of 25-, 21-, and 19-gauge will create pressures of 4, 6, and 8 psi, respectively, when used with a 35-mL syringe.[78]

It is tempting to use higher fluid stream pressures for irrigation as they enhance removal of bacteria and debris from the wound.[75,79] Studies of mechanical irrigation devices used on crushing trauma wounds confirmed that cleaning with a pressure of 70 psi produces significantly more effective removal of bacteria and debris than cleaning with 25 or 50 psi.[80–82] Yet lower pressures are generally desirable in chronic wounds because of the increased risk of dispersing fluid into adjacent wound tissue or along tissue planes with higher pressures.[72–74]

Practice Point

Irrigation at 5 to 15 psi is recommended for heavily exudative wounds. Wound irrigation can be accomplished with a variety of medical tools and specially made devices. A 19-gauge angiocath and a 35-mL syringe is an inexpensive and easy-to-use approach to irrigation.

The wound irrigation fluid stream can be delivered in either a pulsatile or continuous flow pattern. In a randomized clinical trial, pulsed lavage was compared with conventional debridement and Betadine dressing. Findings showed that the pulse lavage resulted in smaller wound size,

less bioburden, and faster healing.[83] Lack of detail in the study methods as to the nature of conventional debridement threatens the generalizability.

An alternate approach to wound irrigation is the whirlpool bath. It cleans the wound by exposing the entire wound bed and surrounding skin to agitating water generated by jets in the sides of the whirlpool tub. Only two studies have investigated the cleaning effectiveness of whirlpool, and these are methodologically confounded with wound irrigation, which was provided at the end of the whirlpool therapy.[84,85] The whirlpool is best suited for use with chronic wounds containing thick slough or necrotic tissue. Caution must also be taken to ensure that the wound doesn't come in close contact with the water jets, since the high pressure they generate could cause further tissue injury.

Topical Agents

Topical antiseptics and antibiotics were out of favor for a period of time because of their toxic effect on cells essential to healing.[86–88] With appreciation of the large population of patients with chronic wounds, the role of biofilm in delayed healing, and the growing number of antibiotic-resistant organisms, treatment perspectives have changed.[9]

Topical agents are currently viewed as essential treatment of critical colonization/biofilm, and wound infection.[3,9,10,19,58,89,90] Topical agents may be in a liquid form, in a gel form, or part of a dressing; studies often evaluate those modes of delivery together. Topical agents are used to kill planktonic organisms, prevent those present on the wound surface and those released with debridement from reattaching, and suppress biofilm reformation. In the initial treatment phase, antiseptics/antibiotics are prescribed empirically until data from DNA analysis (days 4 to 7) come back with the information on the organisms present and their sensitivity to various agents. Schultz et al.[9] recommend changing the antiseptic/antibiotic agent as soon as the sensitivity data become available. Identification of type and susceptibility of organisms in biofilm requires analysis with rapid polymerase chain reaction (PCR).[91] The limitation of this recommendation is the availability of this technology across settings and the knowledge of providers to interpret the data generated.

There remain a number of issues in selecting appropriate treatment. The first major issue is that the effectiveness of the available agents against planktonic and biofilm organisms is not clear.[9,10,92] That leaves the clinician to choose among the agents, based on limited biofilm data and his or her own clinical experience. Selection of an agent also depends on its having low toxicity to cells critical to healing.

The ideal antiseptic has low toxicity and is effective in killing microbes but does not disrupt wound healing. Antiseptics are not site specific and so have a broad impact, affecting both microorganisms and host cells needed for healing and fighting infection.[3,67] An additional consideration is whether the product is effective on

biofilm.[19,89,90,93] Box 7-2 lists antiseptics used to treat increased level of bioburden and biofilm.

BOX 7-2 Antiseptics Used to Control Bioburden and Biofilm

Acetic acid[3,89]

Chlorhexidine[3,89]

EDTA[3,89]

Honey[3,19,58,89]

Iodine[3,19,58,89]

Lactoferrin[3,58]

Methylene blue–gentian violet[3,19]

Mupirocin[3,19]

Polyhexamethylene biguanide[3,89]

Potassium permanganate[3,89]

Silver[3,19,58,89]

Xytol[3,58]

A recent review by Sibbald et al.[92] on topical antimicrobials discusses their toxicity. They propose that low-toxicity agents are saline, tap water, polyhexamethylene biguanide (PHMB), and chlorhexidine, povidone–iodine (PVP-I), and acetic acid. High-toxicity agents include hydrogen peroxide, sodium hypochlorite, quaternary ammonia, and dyes such as scarlet red and proflavine. Topical antimicrobials are formulated from elements such as silver, copper, gold, or zinc, and the specific formulation of the agent is crucial to its effectiveness in reducing bacterial burden without destroying cells essential to healing. The literature on the effect of antimicrobials on bioburden and biofilm is extensive. Many are in vitro studies, so there is question of their effect

on living tissue.[9,30] Also there are a limited number of studies that have in vitro–in vivo comparisons. The study methods vary widely, making it impossible to compare the agents in their ability to reduce or remove biofilm[9,10] Given the state of the science, individual clinicians will need to use existing data and clinical experience to make decisions about which antimicrobial agent to use and how it will be delivered.[9,10] The variability in studies and findings are illustrated below with data about iodine, silver, PHMB, and honey.

Iodine, in its elemental form, is toxic to cells that promote healing. The two most commonly used forms are PVP-I and cadexomer iodine. PVP-I inhibits cellular mechanisms and causes the cells to become denatured and deactivated.[94] It is effective against several bacteria including methicillin-resistant *S. aureus* (MRSA).[95] In vitro studies show that it inhibits *S. epidermidis* and *S. aureus* as well as *Staphylococcus* biofilm formation.[96]

Cadexomer iodine is an alternative iodine product to PVP-I. It is available as an ointment, a dressing, or a powder.[97] It slowly releases iodine from its microspheres while absorbing bacteria.[98,99] An in vitro study of varying concentrations of cadexomer iodine demonstrated that it is nontoxic to human fibroblasts in culture in concentrations of up to 0.45%; the study also showed that chronic wounds treated with cadexomer iodine revealed reepithelialization on biopsy.[99] O'Meara et al.[93] examined the use of antibiotics and antiseptics for venous ulcers. They concluded that some evidence supports the use of cadexomer iodine for treatment of venous leg ulcers. Data do not support the use of honey or silver-based products in these wounds.

Phillips et al.[100] explored the effect of various dressings (cotton gauze, sodium carboxymethylcellulose fiber, calcium alginate, cadexomer beads) and topical agents (iodine, silver, PHMB, honey, ethanol) overtime on *P. aeruginosa* biofilm. The researchers found that time-release silver gel and cadexomer iodine dressings were most effective in reducing mature biofilm while the other dressings reduced biofilm much less at 24 or 72 hours with one exposure. The researchers concluded that there is great variability in the ability of these commonly used dressings to kill mature biofilm. Their efficacy is mediated by time of exposure, number of applications, moisture level,

and agent formulation (sustained release). Subsequently, Fitzgerald et al.[101] examined the effect of cadexomer iodine on biofilms of *P. aeruginosa* and *S. aureus* using an in vitro model. Comparison treatments were various silver products, PHMB, and nonmedicated dressings. Subsequently, cadexomer iodine effects on MRSA were examined in vivo using a mouse wound model. Comparisons were silver dressings and mupirocin. Cadexomer iodine performed best in both models and against *P. aeruginosa*, *S. aureus*, and MRSA mature biofilm.

Topical agents formulated with silver have long been a part of the antibacterial armamentarium. Using the ionized form of silver (Ag+), these agents exert bacteriostatic properties through the action of the silver cation on proteins. The Ag+ binds to proteins in the bacterial cell wall, disrupting its integrity and resulting in death of the cell. Silver-containing wound care products include pure silver, creams, and sustained-release dressings. Research evidence regarding the efficacy of these agents is most substantial for the cream formulations (silver sulfadiazine 2% or 7%). Topical cream formulations have been shown to reduce bacterial density, vascular margination, and migration of inflammatory cells in chronic leg ulcers.[102] The efficacy of silver sulfadiazine cream in reducing bacterial burden in chronic wounds was substantiated in a randomized trial of 45 patients with a single infected PI who were randomly assigned to receive silver sulfadiazine cream, povidone–iodine solution, or saline gauze dressings.[103] In PIs treated with silver sulfadiazine cream, bacterial levels were reduced to 10^5 or less per gram of tissue during the 3-week study period, while only 78.6% of the povidone–iodine solution–treated ulcers, and 63.6% of the saline gauze–treated ulcers achieved these reductions in bacterial levels. In contrast, a review[104] compared silver sulfadiazine with nonantibiotic dressings in venous stasis ulcers. Treatment showed no difference in complete healing at 4 weeks or 1 year.

Treatment of biofilm using silver was studied by Hill et al.[105] in an in vitro model. They tested various silver dressings,[6] iodine dressings,[2] and iodine antiseptics[1] on *Pseudomonas* and *Staphylococcus* biofilm. The iodine containing dressings were effective in killing the biofilm organisms in the 7-day biofilm while topical silver dressings were not.

The ability of agents to kill vegetative bacteria, reduce biofilm, and the toxicity of silver dressings, iodine dressings, and a collagen matrix dressing were evaluated.[106] All the dressing reduced vegetative bacteria counts; however, only the collagen dressing was effective against Pseudomonas aeruginosa. This biofilm effect is thought to be due to the effect of the collagen dressing on proteases, rather than the silver.

Practice Point

Topical antimicrobial therapy is a critical second step in antibiofilm treatment.

Silver frequently is delivered with a silver-impregnated dressing; however, many questions remain regarding the dosage of silver released into the wound and the potential for delays in healing if the silver cation binds to fibroblasts and epithelial cells. Reports of industry-conducted in vitro studies demonstrate that different silver-containing dressings release different amounts of silver over time.[107] There are preliminary indications that the toxic dosage of silver differs depending on whether the fibroblasts and epithelial cells are in a monolayer or in a three-dimensional matrix.[108] The potential adverse consequences in epithelializing wounds were demonstrated in a controlled study of matched pair skin graft donor sites treated with a nonantimicrobial foam dressing or a nanocrystalline silver dressing. Although there were no differences in bacterial counts between the two treatment groups, reepithelialization was significantly slower in the wounds treated with the silver dressing.[109] An evaluation of multiple elements of dressing performance, including silver content, rate of silver release, and antibacterial activity, concluded that the dressing should be selected based on standard clinical parameters rather than on a dressing's silver content or release activity.[110]

PHMB is a broad-spectrum biocide with no known microbial resistance or negative effects on healing. It acts by causing damage to the mitochondria at high concentrations. A randomized controlled study using a PHMB dressing on patients (*n* = 45) with chronic wounds evaluated superficial bioburden, wound-associated pain, and reduction in wound size.[111] At 4 weeks, the PHMB foam dressing reduced bioburden (*P* = 0.016) and pain (*P* = 0.02) and resulted in a greater reduction in wound size (35% vs. 28%).

To et al.[112] reviewed the literature to determine the effect of PHMB as a topical agent for treating chronic wounds. Although the studies included were small (*n* = 10 to 40), data showed that PHMB reduced bacterial count in wounds with superficial bioburden and also reduced wound-related pain.

A Cochrane review by Jull et al.[113] assessed the effects of honey versus other wound dressings and topical agents on healing of wounds. Data showed honey healed infected postoperative wounds more quickly than did antiseptics and gauze, although generally there was poor-quality literature on the effects of honey.

In summary, antiseptics are again appropriate for treatment of chronic wounds. Much of the literature is product/brand specific, and there is lack of data about which antiseptic to use and when. Because the science is limited, clinicians will make treatment decisions based on their understanding of the literature and their clinical experience. While the possibility exists that organisms will become antiseptic resistant, antiseptic resistance has not been seen in vitro to major groups of antiseptics, including PHMB, honey, silver, and iodine.[100]

Systemic Antibiotics

Antibiotic resistance threatens our ability to fight infection, for example, MRSA. There is a need to find alternative approaches to prevent and fight infection, and there has been a growing chorus suggesting approaches. This issue is complicated by the fact that organisms in biofilm can adapt to antibiotics, reducing their metabolism, oxygen consumption, and nutrient requirements, making them much less susceptible to antibiotics.

The step-down/step-up model recommends DNA identification of organisms, and personalized treatment using topical and systemic antimicrobials. Dowd et al.[114] showed that in comparison to standard care where nearly half of patients completely healed, when molecular diagnostic techniques were used to select systemic antibiotics 62% healed, compared to a third group that received molecular driven topical agents that had a healing rate of 90%.

Infected wounds are appropriately treated with systemic antibiotics.[115] While empiric treatment may need to be used initially, this model recommends early identification of personalized antibiotic therapy—both to maximize the effect on the offending organisms and to mitigate the risk of antibiotic resistance. The work by Dowd et al.[114] supports this approach.

One of the questions in care of chronic wounds is whether treatment needs to be wound type specific, for example, venous ulcer, pressure ulcer. To address this issue, the microbiome of nearly 3,000 patients with chronic DFUs, venous ulcers, pressure ulcers, and nonhealing surgical wounds was examined with 16S rDNA sequencing.[116] High levels of *Staphylococcus* (63%) and *Pseudomonas* (25%) were seen as well as a high prevalence of anaerobes. The authors found that neither patient demographics nor wound type affected the microbiome. This suggests that empiric antibiotic therapy need not be dictated by wound type.

The use of a combination of antibiofilm and antimicrobial agents should be used to support healing in chronic wound infections.[10,117] Care should be taken to avoid routine or extended periods of

treatment. Parenteral therapy may be indicated when the infection involves deeper tissue and is accompanied by systemic signs, such as fever, chills, and elevated WBC count. Regardless of the route, the effectiveness of any systemic antibiotic in reducing bacterial burden will be dependent on the adequacy of the patient's peripheral circulation.

Practice Point

The effectiveness of systemic antibiotics is dependent on an adequate blood supply to the wound.

Host Factors and Wound-Specific Treatment

Host factors are evaluated through a comprehensive physical and history. Each patient is evaluated independently to determine that factors fundamental to the wound healing are present.[15,118,119] An adequate blood supply is required as infection occurs more often in ischemic tissues. Local edema needs to be minimized. Oxygen is needed for cellular function. Glucose levels need to be normalized as hyperglycemia inhibits the ability of WBCs to function. Nutritional intake needs to be optimized, so the patient is not catabolic. Underlying diseases need to be identified and the condition of the patient optimized.

Differential diagnosis of chronic wounds may be complex and require thorough evaluation of the patient.[119] Factors specific to the type of wound needs to be managed to maximize available tissue perfusion, for example, diagnostic workup, reduction of edema in venous ulcers, unloading tissues in DFUs, and PIs. Failure to address these basic host factors may contribute to continued proliferation of microorganisms, despite initiation of bioburden/infection-specific treatment.

Practice Point

Care must be focused on placing the patient in the best physical and mental condition to heal.

Adjunctive Therapies

A number of adjunctive therapies are available and need to be considered in early and aggressive treatment, and negative pressure wound therapy (NPWT) is one of them. NPWT is the use of a vacuum dressing to facilitate granulation tissue formation and wound closure.[57] Of the chronic wounds, NPWT had the most positive effect on DFUs, showing higher rates of complete healing, greater reduction in ulcer area, greater reduction in ulcer depth, fewer amputations, and no effect on rate of adverse effects.[120]

The development of NPWT with installation has stimulated renewed interest in NPWT. A prospective study of the effect of a surgical debridement followed by NPWT with an irrigant of 0.125% sodium hypochlorite on biofilm in patients with chronic wounds showed a 48% reduction in quantitative biofilm-protected bacteria while the control had a mean 14% increase.[121] Further work is needed with larger samples to confirm these findings.

Collagen dressings differ in composition, and each type of dressing has different modes of action and inactivates different proteases. Those composed of natural collagen (not denatured or reconstructed) are beneficial because they may both be able to reestablish balance (reduce MMPs) and promote healing. When used early in the treatment cycle, they buffer excess MMPs; when used later in the repair process, they may be absorbed into the wound and help with collagen organization, migration, and proliferation.[30]

Agents targeting the exopolysaccharide (EPS) are being explored as a means to release the trapped biofilm organisms. Freeing the organisms from EPS would allow the full effect of the patient's immune system as well as antimicrobials to fight infection. More work is needed to demonstrate their effects.[115]

Quorum sensing inhibitors also are being explored. They are designed to interfere with biofilm production and/or matrix development. The goal is to increase the susceptibility of the organisms in the biofilm to antibiotics, thus reducing antibiotic doses.

Quorum sensing is an interesting and unique characteristic of biofilm, and further work is needed in this area.[115]

Targeted therapy based on molecular microbiology diagnostics is another antibiofilm strategy. Strong support for the use of molecular diagnosis is provided by a retrospective cohort study that compared complete healing when in a large cohort of patients (*n* = 1,378) managed for bioburden.[114] Empirically prescribed antibiotics resulted in 48.5% complete healing, 62.4% when systemic antibiotics were prescribed based on molecular diagnosis and 90.4% when topical agents (including antibiotics) were prescribed based on molecular diagnostics.

Summary

Most of our understanding of wound infection has been derived from the study of acute wounds. As wound healing science has evolved, it has become clear that chronic wounds are distinctly different environments where host resistance has been overwhelmed by bacterial burden. The classic signs and symptoms of infection are well recognized. However, they're based on assessments made of acute wounds and aren't valid in the chronic wound. While indicators of chronic wound infection remain ambiguous, substantial evidence shows that debridement of necrotic tissue and biofilm in the wound bed is an essential first step to reducing bacterial burden. Early and aggressive treatment of those at risk or with biofilm is proposed. Debridement, use of topical antiseptics/systemic antibiotics, ongoing evaluation of healing and inflammation, optimizing host factors for healing, addressing the underlying etiology of the wound, and implementing adjunct therapies are needed to address biofilm.

PATIENT SCENARIO

Clinical Data

Mr. S is a 73-year-old well-developed, well-nourished white male who presents today with a nonhealing wound on the lateral aspect of the malleolus.

He reports that the wound began about 2 months ago when he inadvertently hit the ankle with a tool while working in the garden. Since then he has kept the area covered, but it seems to be increasing in size and drains copious amounts of serous fluid that is malodorous and limits his ability to go out of the house.

Clinical examination of the wound show the wound is 3 cm × 6 cm × 0.6 cm. About half is covered with yellow slough, and it is heavily exudative. The ulcer is malodorous and painful. No edema is appreciated. The area is not warm, and there are no satellite wounds. His history is negative for diabetes and pulmonary disease. He

experienced a pulmonary embolism 5 years ago and is on Coumadin. He takes no other prescription medications.

Case Discussion

Analysis of the history and the signs and symptoms show Mr. S's wound is experiencing critical colonization/biofilm. Having confirmed the presence of adequate circulation and a normal nutritional state, the wound was sharply debrided and cadexomer iodine applied topically, covered with a foam dressing. Local treatment prescribed included daily cleansing with normal saline using a 35-mL syringe and 19-gauge angiocath followed by topical application of the cadexomer covered with a foam dressing. The patient was seen in a week, and the odor and exudate had abated. He again underwent sharp debridement, and continued the cadexomer iodine. When seen in clinic 3 weeks after his initial visit, the wound bed was granulating, and the wound size had decreased slightly (2.9 cm × 5.8 cm × 0.7 cm).

Show What You Know

1. **What is the current thinking about the use of topical antiseptics in chronic wounds?**
   A. They should never be used because biofilm organisms are resistant to them.
   B. They should never be used because they injure cells needed for wound healing.
   C. They should be used because they have site-specific action on bacteria.
   D. They should be used because they kill planktonic organisms and enhance wound closure.

2. **Which of the following most accurately indicates infection in acute wounds?**
   A. A tissue biopsy showing large amounts of *Staphylococcus aureus*
   B. A quantitative culture of tissue showing 1,000 organisms/g of tissue
   C. A semiquantitative culture showing 4+ organisms present
   D. A tissue biopsy with less than 10^5 CFU organisms present

3. **Biofilm in chronic wounds:**
   A. is uniformly distributed across a chronic wound.
   B. is diagnosed with the presence of slough.
   C. contains organisms that are easily treated with systemic antibiotics.
   D. cannot be seen with the naked eye.

4. **The most effective debridement of chronic wound is:**
   A. use of topical enzymes for up to 4 weeks.
   B. continuous use of autolytic debridement with agents such as hydrogels.
   C. weekly sharp debridement.
   D. larvae (maggot) therapy for 2 weeks

Answers to these questions are found on pages 682 and 683.

References

1. Sanford, J.A., Gallo, R.L. Functions of the Skin Microbiota in Health and Disease. Seminars in Immunology 25(5):370-7, 2013.
2. Grice, E.A., Segre, J.A. The Human Microbiome: Our Second Genome. Annual Review of Genomics and Human Genetics 13:151-70, 2013.
3. Percival, S., Cutting K., eds. Microbiology of Wounds. Boca Raton, FL: CRC Press, Taylor & Francis Group, 2010.
4. Gottrup, F., Apelqvist, J., Bjansholtm, T., et al. EWMA Document: Antimicrobials and Non-Healing Wounds: Evidence, Controversies and Suggestions. Journal of Wound Care 22(5 Suppl):S1-89, 2013.
5. Percival, S.L., Hill, K.E., Williams, D.W., et al. A Review of the Scientific Evidence for Biofilms in Wounds. Wound Repair and Regeneration 20(5):647-57, 2012.
6. Metcalf, D.G., Bowler, P.G., Hurlow, J. A Clinical Algorithm for Wound Biofilm Identification. Journal of Wound Care 23(3):137-8, 140-42, 2014.
7. Attinger, C., Wolcott, R. Clinically Addressing Biofilm in Chronic Wounds. Advances in Wound Care 1(3):127-32, 2012.
8. Thomson, C.H. Biofilms: Do They Affect Wound Healing? International Wound Journal 8(1):63-7, 2011.
9. Schultz, G., Bjarnsholt, T., Cutting, K., et al. Global Wound Biofilm Expert Panel. Consensus Guidelines for the Identification and Treatment of Biofilms in Chronic Nonhealing Wounds. Wound Repair and Regeneration 25(5):744-57, 2017.
10. Snyder, R.J., Bohn, G., Hanft, J., et al. Wound Biofilm: Current Perspectives and Strategies on Biofilm Disruption and Treatments. Wounds 29(6):S1-17, 2017.
11. del Pozo, J.L., Patel, R. The Challenge of Treating Biofilm-Associated Bacterial Infections. Clinical Pharmacology & Therapeutics 82(2):204-9, 2007.
12. Kim, P.D., Xia-Juan, X., Crump, K.E., et al. Toll-Like Receptor 9-Mediated Inflammation Triggers Alveolar Bone Loss in Experimental Murine Periodontitis. Infection and Immunity 83(7):2992-3002, 2015.
13. Sun, Y., Zhou, B., Wang, C., et al. Biofilm Formation and Toll-like Receptor 2, Toll-like Receptor 4, and NF-kappaB Expression in Sinus Tissues of Patients with Chronic Rhinosinusitis. American Journal of Rhinology & Allergy 26(2):104-9, 2012.
14. James, G.A., Swogger, E., Wolcott, R., et al. Biofilms in Chronic Wounds. Wound Repair and Regeneration 16(1):37-44, 2008.
15. Sibbald, R.G., Woo, K., Ayello, E.A. Increased Bacterial Burden and Infection: The Story of NERDS and STONES. Advances in Skin & Wound Care 19(8): 447-61, quiz 461-63, 2006.
16. Wolcott, R.D., Rumbaugh, K.P., James, G., et al. Biofilm Maturity Studies Indicate Sharp Debridement Opens a Time-Dependent Therapeutic Window.

Journal of Wound Care 19(8):320-8, 2010.
17. Cutting, K.F., Harding, K.G. Criteria for Identifying Wound Infection. Journal of Wound Care 3(4):198-201, 1994.
18. Gardner, S.E., Frantz, R.A., Doebbeling, B.N. The Validity of the Clinical Signs and Symptoms Used to Identify Localized Chronic Wound Infection. Wound Repair and Regeneration 9(3):178-86, 2001.
19. Jones, C.E., Kennedy, J.P. Treatment Options to Manage Wound Biofilm. Advances in Wound Care 1(3):120-6, 2012.
20. Wilgus, T.A., Roy, S., McDaniel, J.C. Neutrophils and Wound Repair: Positive Actions and Negative Reactions. *Advances in Wound Care* 2(7):379-88, September 2013.
21. Knighton, D.R., Halliday, B., Hunt, T.K. Oxygen as an Antibiotic. A Comparison of the Effects of Inspired Oxygen Concentration and Antibiotic Administration on In Vivo Bacterial Clearance. Archives of Surgery 121(2):191-5, 1986.
22. Jensen, P.Ø., Bjarnsholt, T., Phipps, R., et al. Rapid Necrotic Killing of Polymorphonuclear Leukocytes is Caused by Quorum-Sensing-Controlled Production of Rhamnolipid by Pseudomonas aeruginosa. Microbiology 153(Pt 5):1329-38, 2007.
23. Grice, E.A., Segre, J.A. Interaction of the Microbiome with the Innate Immune Response in Chronic Wounds. Advances in Experimental Medicine & Biology 946: 55-68, 2012.
24. Mu, Y., Edwards, J.R., Horan, T.C., et al. Improving Risk-Adjusted Measures of Surgical Site Infection for the National Healthcare Safety Network. Infection Control and Hospital Epidemiology 32(10):970-86, 2011.
25. Reddy, M., Gill, S.S., Wu, W., et al. Does this Patient Have an Infection of a Chronic Wound? JAMA 307(6):605-11, 2012.
26. American College of Surgeons: Committee on the Control of Surgical Infections. Manual on Control of Infection in Surgical Patients. Philadelphia, PA: Lippincott Williams & Wilkins, 1986.
27. Robson, M.C., Heggers, J.P. Surgical Infection. II. The Beta-Hemolytic Streptococcus. Journal of Surgical Research 9(5):289-92, 1969.
28. Davies, D. Understanding Biofilm Resistance to Antibacterial Agents. Nature Reviews Drug Discovery 2(2):114-22, 2003.
29. Berríos-Torres, S.I., Umscheid, C.A., Bratzler, D.W., et al. Healthcare Infection Control Practices Advisory Committee. Centers for Disease Control and Prevention Guideline for the Prevention of Surgical Site Infection. JAMA Surgery 152(8):784-91, 2017.
30. Bohn, G.A., Schultz, G.S., Liden, B.A., et al. Proactive and Early Aggressive Wound Management: A Shift in Strategy Developed by a Consensus Panel Examining the Current Science, Prevention, and Management of Acute and Chronic Wounds. Wounds 29(11):S37-42, 2017.
31. Horan,T.C., Gaynes, R.P., Martone, W.J., et al. CDC Definitions of Nosocomial Surgical Site Infections, 1992: A Modification of CDC Definitions of Surgical

Wound Infections. Infection Control and Hospital Epidemiology 20(5):271-4, 1992.
32. Cutting, K.F. Identification of Infection in Granulating Wounds by Registered Nurses. Journal of Clinical Nursing 7(6):539-46, 1998.
33. Gardner, S.E., Frantz, R.A., Park, H., et al. The inter-rater Reliability of the Clinical Signs and Symptoms Checklist in Diabetic Foot Ulcers. Ostomy/Wound Management 53(1):46-51, 2007.
34. Gardner, S.E., Hillis, S.L., Frantz, R.A. Clinical Signs of Infection in Diabetic Foot Ulcers with High Microbial Load. Biological Research for Nursing 11(2):119-28, 2009.
35. Lipsky, B.A., Berendt, A.R., Deery, H.G., et al. Infectious Diseases Society of America. Diagnosis and Treatment of Diabetic Foot Infections Clinical Infectious Diseases 39(7):885-910, 2004.
36. Woo, K.Y., Sibbald, R.G. A Cross-Sectional Validation Study of Using NERDS and STONES to Assess Bacterial Burden. Ostomy/Wound Management 55(8): 40-8, 2009.
37. Fazli, M., Bjarnsholt, T., Kirketerp-Moller, K., et al. Nonrandom Distribution of Pseudomonas aeruginosa and Staphylococcus aureus in Chronic Wounds. Journal of Clinical Microbiology 47(12):4084-9, 2009.
38. Cutting, K.F., Harding, K. Clinical Identification of Wound Infection: A Delphi Approach in EWMA Position Document—Identifying Criteria for Wound Infection. London, UK: MEP Ltd., 2005:6-9.
39. Robson, M.C., Heggers, J.P. Bacterial Quantification of Open Wounds. Military Medicine 134, 19-24: 1969.
40. Krizek, T.J., Robson, M.C., Kho, E. Bacterial Growth and Skin Graft Survival. Surgical Forum 18:518-9, 1967.
41. Robson, M.C., Lea, C.E., Dalton, J.B., et al. Quantitative Bacteriology and Delayed Wound Closure. Surgical Forum 19:501-2, 1968.
42. Kallstrom, G. Are Quantitative Bacterial Wound Cultures Useful? Journal of Clinical Microbiology 52(8):2753-6, 2014.
43. Gardner, S.E., Frantz, R.A., Saltzman, C.L., et al. Diagnostic Validity of Three Swab Techniques for Identifying Chronic Wound Infection. Wound Repair and Regeneration 14(5):548-57, 2006.
44. Lee, P.C., Turnidge, J., McDonald, P.J. Fine-Needle Aspiration Biopsy in Diagnosis of Soft Tissue Infections. Journal of Clinical Microbiology 22(1):80-3, 1985.
45. Dow, G., Browne, A., Sibbald, R.G. Infection in Chronic Wounds: Controversies and Treatment. Ostomy/Wound Management 45(8):23-40, 1999.
46. Rudensky, B., Lipschits, M., Isaacsohn, M., et al. Infected Pressure Sores: Comparison of Methods for Bacterial Identification. Southern Medical Journal 85(9):901-3, 1992.
47. Levine, N.S., Lindberg, R.B., Mason, A.D., Jr., et al. The Quantitative Swab Culture and Smear: A Quick, Simple Method for Determining the Number of

Viable Aerobic Bacteria on Open Wounds. Journal of Trauma 16(2):89-94, 1976.
48. Cooper, R., Lawrence, J.C. The Isolation and Identification of Bacteria from Wounds. Journal of Wound Care 5(7):335-40, 1996.
49. Bill, T.J., Ratliff, C.R., Donovan, A.M., et al. Quantitative Swab Culture Versus Tissue Biopsy: A Comparison in Chronic Wounds. Ostomy/Wound Management 47(1):34-7, 2001.
50. Basak, S., Dutta, S.K., Gupta, S., et al. Bacteriology of Wound Infection: Evaluation by Surface Swab and Quantitative Full Thickness Wound Biopsy Culture. Journal of the Indian Medical Association 90(2):33-4, 1992.
51. Copeland-Halperin, L.R., Kaminsky, A.J., Bluefeld, N., et al. Sample Procurement for Cultures of Infected Wounds: A Systematic Review. Journal of Wound Care 25(4):S4-6, S8-10, 2016.
52. Haalboom, M., Blokhuis-Arkes, M.H.E., Beuk, R.J., et al. Culture Results from Wound Biopsy Versus Wound Swab: Does It Matter for the Assessment of Wound Infection? Clinical Microbiology and Infection 2018, pii, S1198-743X(18)30586-X, 2018.
53. Wu, Y.K., Cheng, N.C., Cheng, C.M. Biofilms in Chronic Wounds: Pathogenesis and Diagnosis. Trends in Biotechnology, pii, S0167-7799(18)30307-X, 2018.
54. Nakagami, G., Schultz, G., Gibson, D.J., et al. Biofilm Detection by Wound Blotting Can Predict Slough Development in Pressure Ulcers: A Prospective Observational Study. Wound Repair Regeneration 25(1):131-8, 2017.
55. Schultz, G., Wolcott, R.D. Wound Infection and Diagnostics in Practice: What Is Emerging? International Wound Journal 4(1):4-6, 2013.
56. Schultz, G.S., Sibbald, R.G., Falanga, V., et al. Wound Bed Preparation: A Systematic Approach to Wound Management. Wound Repair & Regeneration 1:S1-28, 2003.
57. Leaper, D.J., Schultz, G., Carville, K., et al. Extending the TIME Concept: What Have We Learned in the Past 10 Years? International Wound Journal 9(Suppl 2): 1-19, 2012.
58. Wolcott, R.D., Rhoads, D.D. A Study of Biofilm-Based Wound Management in Subjects with Critical Limb Ischaemia. Journal of Wound Care 17(4):145-48, 150-52, 154-55, 2008.
59. Howell, R.S., Gorenstein, S., Castellano, M., et al. Wound Care Center of Excellence: Guide to Operative Technique for Chronic Wounds. Journal of the American College of Surgeons 226(2):e7-17, 2018.
60. Bekara, F., Vitse, J., Fluierarum, S., et al. New Techniques for Wound Management: A Systematic Review of Their Role in the Management of Chronic Wounds. Archives of Plastic Surgery 45(2):102-10, 2018.
61. Schultz, G.S., Woo, K., Weir, D., et al. Effectiveness of a Monofilament Wound Debridement Pad at Removing Biofilm and Slough: Ex Vivo and Clinical Performance. Journal of Wound Care 27(2):80-90, 2018.

62. Pritchard, D.I., Čeřovský, V., Nigam, Y., et al. TIME Management by Medicinal Larvae. International Wound Journal 13(4):475-84, 2016.
63. Dumville, J.C., Worthy, G., Bland, J.M., et al. Larval Therapy for Leg Ulcers (VenUS II): Randomised Controlled Trial. BMJ 338:b773, 2009.
64. Opletalová, K., Blaizot, X., Mourgeon, B., et al. Maggot Therapy for Wound Debridement: A Randomized Multicenter Trial. Archives of Dermatology 148(4): 432-8, 2012.
65. Mudge, E., Price, P., Walkley, N., et al. A Randomized Controlled Trial of Larval Therapy for the Debridement of Leg Ulcers: Results of a Multicenter, Randomized, Controlled, Open, Observer Blind, Parallel Group Study. Wound Repair & Regeneration 22(1): 43-51, 2014.
66. Sun, X., Jiang, K., Chen, J., et al. A Systematic Review of Maggot Debridement Therapy for Chronically Infected Wounds and Ulcers. International Journal of Infectious Diseases 25:32-7, 2014.
67. World Union of Wound Healing Societies (WUWHS). Principles of Best Practice: Wound Infection in Clinical Practice. An International Consensus. London, UK: MEP Ltd. Available at www.mepltd.co.uk. 2008.
68. Fernandez, R., Griffiths, R. Water for Wound Cleansing. Cochrane Database of Systematic Reviews (2):CD003861, 2012.
69. Foresman, P.A., Payne, D.S., Becker, D., et al. A Relative Toxicity Index for Wound Cleansers. Wounds 5:226-31, 1993.
70. Wilson, J.R., Mills, J.G., Prather, I.D., et al. A Toxicity Index of Skin and Wound Cleansers Used on In Vitro Fibroblasts and Keratinocytes. Advances in Skin & Wound Care 18(7):373-8, 2005.
71. Rani, S.A., Hoon, R., Najafi, R.R., et al. The In Vitro Antimicrobial Activity of Wound and Skin Cleansers at Nontoxic Concentrations. Advances in Skin & Wound Care 27(2):65-9, 2014.
72. Bhaskar, S.N., Cutright, D.E., Gross, A. Effect of Water Lavage on Infected Wounds in the Rat. Journal of Periodontology 40(11):671-2, 1969.
73. Carlson, H.C., Briggs, R.L., Green, V.A., et al. Effect of Pressure and Tip Modification on the Dispersion of Fluid Throughout Cells and Tissues During the Irrigation of Experimental Wounds. Oral Surgery, Oral Medicine, and Oral Pathology 32(2):347-55, 1971.
74. Wheeler, C.B., Rodeheaver, G.T., Thacker, J.G., et al. Side Effects of High Pressure Irrigation. Surgery, Gynecology & Obstetrics 143(5):775-8, 1976.
75. Rodeheaver, G.T., Pettry, D., Thacker, J.G., et al. Wound Cleansing by High Pressure Irrigation. Surgery, Gynecology & Obstetrics 141(3):357-62, 1975.
76. Longmire, A.W., Broom, L.A., Burch, J. Wound Infection Following High-Pressure Syringe and Needle Irrigation. American Journal of Emergency Medicine 5(2):179-81, 1987.
77. Stevenson, T.R., Thacker, J.G., Rodeheaver, G.T., et al. Cleansing the Traumatic Wound by High Pressure Syringe Irrigation. JACEP 5(1):17-21, 1976.

78. Singer, A.J., Hollander, J.E., Subramanian, S., et al. Pressure Dynamics of Various Irrigation Techniques Commonly Used in the Emergency Department. Annals of Emergency Medicine 24(1):36-40, 1994.
79. Madden, J., Edlich, R.E., Schauerhamer, R. Application of Principles of Fluid Dynamics to Surgical Wound Irrigation. Current Topics in Surgical Research 3:85-93, 1971.
80. Grower, M.F., Bhaskar, S.N., Horan, M.J., et al. Effect of Water Lavage on Removal of Tissue Fragments from Crush Wounds. Oral Surgery, Oral Medicine, and Oral Pathology 33(6):1031-6, 1972.
81. Green, V.A., Carlson, H.C., Briggs, R.L. A Comparison of the Efficacy of Pulsed Mechanical Lavage with that of Rubber-Bulb Syringe Irrigation in Removal of Debris from Avulsive Wounds. Oral Surgery, Oral Medicine, and Oral Pathology 32(1):158-64, 1971.
82. Stewart, J.L., Carlson, H.C., Briggs, R.L., et al. The Bacteria-Removal Efficiency of Mechanical Lavage and Rubber-Bulb Syringe Irrigation in Contaminated Avulsive Wounds. Oral Surgery, Oral Medicine, and Oral Pathology 31(6):842-8, 1971.
83. Shetty, R., Barreto, E., Paul, K.M. Suction Assisted Pulse Lavage: Randomized Controlled Studies Comparing Its Efficacy with Conventional Dressings in Healing of Chronic Wounds. International Wound Journal 11(1):55-63, 2014.
84. Bohannon, R.W. Whirlpool Versus Whirlpool Rinse for Removal of Bacteria from a Venous Stasis Ulcer. Physical Therapy 62(3):304-8, 1982.
85. Niederhuber, S.S., Stribley, R.F., Koepke, G.H. Reduction of Skin Bacterial Load with Use of the Therapeutic Whirlpool. Physical Therapy 55(5):482-6, 1975.
86. Lineaweaver, W., Howard, R., Soucy, D., et al. Topical Antimicrobial Toxicity. Archives of Surgery 120(3):267-70, 1985.
87. Viljanto, J. Disinfection of Surgical Wounds Without Inhibition of Normal Wound Healing. Archives of Surgery 115:253-6, 1980.
88. Cooper, M.L., Laxer, J.A., Hansbrough, J.F. The Cytotoxic Effects of Commonly Used Topical Antimicrobial Agents on Human Fibroblasts and Keratinocytes. Journal of Trauma 31(6):775-82; discussion 782-84, 1991.
89. Vowden, P., Vowden, K., Carville, K. Antimicrobial Dressings Made Easy. Wounds International 2(1):1-6, 2014.
90. Ammons, M.C. Anti-Biofilm Strategies and the Need for Innovations in Wound Care. Recent Patents on Anti-Infective Drug Discovery 5(1):10-7, 2010.
91. Wolcott, R.D., Ehrlich, G.D. Biofilms and Chronic Infections. JAMA 299(22):2682-4, 2008.
92. Sibbald, R.G., Elliott, J.A., Verma, L., et al. Update: Topical Antimicrobial Agents for Chronic Wounds. Advances in Skin & Wound Care 30(10), 438-50, 2017.
93. O'Meara, S., Richardson, R., Lipsky, B.A. Topical and Systemic Antimicrobial Therapy for Venous Leg Ulcers. JAMA 311(24):2534-5, 2014.

94. Kanagalingam, J., Feliciano, R., Hah, J.H., et al. Practical Use of Povidone-Iodine Antiseptic in the Maintenance of Oral Health and in the Prevention and Treatment of Common Oropharyngeal Infections. International Journal of Clinical Practice 69(11):1247-56, 2015.
95. Bigliardi, P.L., Alsagoff, S.A.L., El-Kafrawi, H.Y., et al. Povidone Iodine in Wound Healing: A Review of Current Concepts and Practices. International Journal of Surgery 44:260-8, 2017.
96. Oduwole, K.O., Glynn, A.A., Molony, D.C., et al. Anti-Biofilm Activity of Sub-Inhibitory Povidone-Iodine Concentrations Against Staphylococcus epidermidis and Staphylococcus aureus. Journal of Orthopaedic Research 28(9):1252-6, 2010.
97. Percival, S.L., Finnegan, S., Donelli, G., et al. Antiseptics for Treating Infected Wounds: Efficacy on Biofilms and Effect of pH. Critical Reviews in Microbiology 42(2):293-309, 2016.
98. Stadelmann, W.K., Digenis, A.G., Tobin, G.R. Impediments to Wound Healing. American Journal of Surgery 176(2A Suppl):39S-47S, 1998.
99. Zhou, L.H., Nahm, W.K., Badiavas, E., et al. Slow Release Iodine Preparation and Wound Healing: In Vitro Effects Consistent with Lack of In Vivo Toxicity in Human Chronic Wounds. British Journal of Dermatology 146(3):365-74, 2002.
100. Phillips, P.L., Yang, Q., Davis, S., et al. Antimicrobial Dressing Efficacy Against Mature Pseudomonas aeruginosa Biofilm on Porcine Skin Explants. International Wound Journal 12(4):469-83, 2015.
101. Fitzgerald, D.J., Renick, P.J., Forrest, E.C., et al. Cadexomer Iodine Provides Superior Efficacy Against Bacterial Wound Biofilms In Vitro and In Vivo. Wound Repair and Regeneration 25(1):13-24, 2017.
102. Fumal, I., Braham, C., Paquet, P., et al. The Beneficial Toxicity Paradox of Antimicrobials in Leg Ulcer Healing Impaired by a Polymicrobial Flora: A Proof-of-Concept Study. Dermatology 204(Suppl. 1): 70-4, 2002.
103. Kucan, J.O., Robson, M.C., Heggers, J.P., et al. Comparison of Silver Sulfadiazine, Povidone-iodine and Physiologic Saline in the Treatment of Chronic Pressure Ulcers. Journal of American Geriatrics Society 29(5):232-5, 1981.
104. Miller, A.C., Rashid, R.M., Falzon, L., et al. Silver Sulfadiazine for the Treatment of Partial-Thickness Burns and Venous Stasis Ulcers. Journal of the American Academy of Dermatology 66(5):e159-65, 2012.
105. Hill, K.E., Malic, S., McKee, R., et al. An In Vitro Model of Chronic Wound Biofilms to Test Wound Dressings and Assess Antimicrobial Susceptibilities. Journal of Antimicrobial Chemotherapy 65(6):1195-206, 2010.
106. Bourdillon, K.A., Delury, C.P., Cullen, B.M. Biofilms and Delayed Healing—An In Vitro Evaluation of Silver- and Iodine-Containing Dressings and Their Effect on Bacterial and Human Cells. International Wound Journal 14(6):1066-75, 2017.
107. Ovington, L.G. The Truth About Silver. Ostomy/Wound Management 50(9A Suppl):1S-10S, 2004.

108. Poon, V.K.M., Burd, P. In Vitro Cytotoxicity of Silver: Implications for Clinical Wound Care. Burns 30(2):140-7, 2004.
109. Innes, M.E., Umraw, N., Fish, J.S., et al. The Use of Silver Coated Dressings on Donor Site Wounds: A Prospective, Controlled Matched Pair Study. Burns 27(6):621-7, 2001.
110. Parsons, D., Bowler, P.G., Myles, V., et al. Silver Antimicrobial Dressings in Wound Management: A Comparison of Antibacterial, Physical, and Chemical Characteristics. Wounds 17(8):222-32, 2005.
111. Sibbald, R.G., Coutts, P., Woo, K.Y. Reduction of Bacterial Burden and Pain in Chronic Wounds Using a New Polyhexamethylene Biguanide Antimicrobial Foam Dressing-Clinical Trial Results. Advances in Skin & Wound Care 24(2):78-84, 2011.
112. To, E., Dyck, R., Gerberm S., et al. The Effectiveness of Topical Polyhexamethylene Biguanide (PHMB) Agents for the Treatment of Chronic Wounds: A Systematic Review. Surgical Technology International 29:45-51, 2016.
113. Jull, A.B., Cullum, N., Dumville, J.C., et al. Honey as a Topical Treatment for Wounds. Cochrane Database of Systematic Reviews (3):CD005083, 2015.
114. Dowd, S.E., Wolcott, R.D., Kennedy, J., et al. Molecular Diagnostics and Personalised Medicine in Wound Care: Assessment of Outcomes. Journal of Wound Care 20(5):232, 234-9, 2011.
115. Gompelman, M., van Asten, S.A., Peters, E.J. Update on the Role of Infection and Biofilms in Wound Healing: Pathophysiology and Treatment. Plastic and Reconstructive Surgery 138(3 Suppl):61S-70S, 2016.
116. Wolcott, R.D., Hanson, J.D., Rees, E.J., et al. Analysis of the Chronic Wound Microbiota of 2,963 Patients by 16S rDNA Pyrosequencing. Wound Repair and Regeneration 24(1):163-74, 2018.
117. Bowler, P.G. Antibiotic Resistance and Biofilm Tolerance: A Combined Threat in the Treatment of Chronic Infections. Journal of Wound Care 27(5): 273-7, 2018.
118. Sibbald, R.G., Goodman, L., Woo, K.Y., et al. Special Considerations in Wound Bed Preparation 2011: An Update©. Advances in Skin & Wound Care 24(9):415-36, quiz 437-38, 2011.
119. Morton, L.M., Phillips, T.J. 2016 Wound Healing and Treating Wounds: Differential Diagnosis and Evaluation of Chronic Wounds. Journal of the American Academy of Dermatology 74(4):589-605, quiz 605-6.
120. Liu, S., He, C.Z., Cai, Y.T., et al. Evaluation of Negative-Pressure Wound Therapy for Patients with Diabetic Foot Ulcers: Systematic Review and Meta-Analysis. Therapeutics and Clinical Risk Management 13:533-44, 2017.
121. Yang, C., Goss, S.G., Alcantara, S., et al. Effect of Negative Pressure Wound Therapy With Instillation on Bioburden in Chronically Infected Wounds. Wounds 29(8), 240-6, 2017.

8

Wound Debridement

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Objectives

After completing this chapter, you'll be able to:

- assess the purpose of wound debridement
- evaluate criteria for *not* debriding a necrotic wound
- describe types of debridement, including sharp/surgical, conservative surgical, mechanical, maggot, enzymatic, and autolytic
- compare the advantages and disadvantages of each debridement type
- select the most appropriate debridement method depending on patient preference, clinician expertise, and healthcare system resources.

Speeding the Healing Process

Debridement is an important component of the wound bed preparation (WBP) management model.[1–4] After considering the cause of the wound and patient-centered concerns, debridement is a necessary step in local wound care.[1,2,4] Debridement is defined as *the removal of necrotic tissue, exudate, bacteria, and metabolic waste from a wound in order to improve or facilitate the healing process.*[3,4] Accumulation of necrotic tissue usually results from poor blood supply, a prolonged inflammatory process, bacterial damage, or an untreated cause of the wound (e.g., uncontrolled edema leading to increased interstitial pressure or other mechanical, chemical, or traumatic injury). In otherwise healthy persons with wounds, natural debridement often keeps pace with the accumulation of dying tissue. If the host resistance is impaired by poor nutrition, continued pressure damage, or other comorbidities including uncontrolled high blood sugar in a person with diabetes, medical intervention is required to facilitate wound healing.

The primary purpose of debridement is to reduce or remove dead and necrotic tissue in a healable wound because this tissue is a proinflammatory stimulus and a culture medium for bacterial growth.[1,3,4] The removal of dead and necrotic tissue is necessary to reduce the biological burden of the wound (e.g., planktonic bacteria or biofilms) in order to control critical colonization (local infection) on the wound surface and to decrease the likelihood of deep and surrounding wound infection.[4] Debridement allows the practitioner to visualize the sides and base of a wound more accurately to determine whether viable tissue is present. (Keep in mind that a pressure injury that is covered with necrotic tissue cannot be numerically staged until debridement is completed.) Necrotic tissue that is not removed not only impedes wound healing but also can result in spread of bacterial damage to deeper tissue, causing surrounding cellulitis, osteomyelitis, and the possibility of septicemia, preventable limb amputation, or death. By removing necrotic tissue, debridement creates an acute wound within a chronic wound, restoring nutrient supply from the

underlying circulation and facilitating optimal available oxygen delivery to the wound site.[4,5]

For a wound to optimally heal, it must have a microenvironment relatively free from the nonviable tissue that serves as a bacterial culture medium to increase bacterial metabolism and proliferation.[6] Oxygen is a primary requirement for this energy-dependent metabolic process to occur. The production of free radicals crucial to wound healing facilitates this process by killing bacteria and promoting the proliferation of both fibroblasts and epithelial cells. However, bacteria that are present in hypoxic conditions (anaerobes, facultative aerobes, and bacteria in biofilms) compete with healing tissue for nutrients and produce an inflammatory environment with exotoxins and endotoxins that damage newly generated, mature cells. This setting of hypoxia and bacterial inflammatory damage disrupts the fundamental wound healing process of fibroblast migration into the extracellular matrix.

For optimal healing, fibroblasts excrete procollagen fibers that are assembled in the extracellular space (with the aid of vitamin C) into a collagen matrix that forms the building blocks of granulation tissue. This process allows a chronic wound stuck in the destructive abnormally prolonged inflammatory stage to move to the proliferative stage, promoting new tissue formation and laying the foundation for healing with the necessary recruitment of collagen depositing fibroblasts.[6]

Leukocytes—primarily polymorphonuclear cells—are the primary cells of the acute wound healing inflammatory process. They enter the wound and aid in the removal of debris (devitalized tissue, bacteria, foreign material). Local enzymes (metalloproteases, elastases, or collagenases) also help to dissolve and remove devitalized tissue. Because collagen comprises approximately 75% of the skin's dry weight, the overall endogenous collagenase is considered to be one of the main regulators of wound healing–related tissue remodeling. This remodeling is part of the final restructuring of the healing process.

Tissue injury is followed by a clotting stage and then the inflammatory stage to remove tissue debris. After wound debris is

removed, macrophages are recruited; they, in turn, recruit fibroblasts, which deposit collagen and fill the wound space with scar tissue. An acute wound of reasonable size with adequate blood supply for healing and essential nutrients generally results in a “healed” closed wound within 14 days—but this doesn’t represent the total healing process. Remodeling, or maturation, typically takes another 4 weeks, making the total healing process about 6 weeks, but the further increase in tensile strength can take a year or even longer. Other factors may be involved in the healing process as well. For example, a wound that appears in an area of rich blood supply (e.g., scalp) will heal faster than a wound in an area with a lesser blood supply (e.g., sacrum). Abnormal collagen breakdown and new collagen buildup occur in a balanced growth pattern resulting in a normal-appearing scar.

However, excess collagen can form a hypertrophic scar or keloid. A hypertrophic scar represents disordered collagen within the scar tissue margins of a healed wound. A keloid extends beyond the wound scar margin with a greater disorganization of collagen fibers.

Identifying Necrotic Tissue

Dead or necrotic tissue may be loose and moist or dry and firm. This tissue is identified by its soft loose moist, yellow, green, or gray appearance and may become hard thick and leathery with a dry black eschar (Fig. 8-1). Oxygen and nutrients can't penetrate the portion of a wound composed of necrotic tissue. Dead tissue is the breeding ground for bacterial proliferation, and the eschar may mask an underlying abscess.[7] Necrotic tissue that is soft, moist, stringy, and yellow is referred to as slough (devitalized/avascular) tissue.[8–11] While some clinical experts have questioned whether slough is in fact tissue or an inflammatory by-product,[10] the European Pressure Ulcer Advisory Panel (EPUAP), National Pressure Injury Advisory Panel (NPIAP), and Pan Pacific Pressure Injury Alliance (PPPIA) 2019 Prevention and Treatment of Pressure Ulcers/Injuries Clinical Practice Guideline: The International Guideline did define slough as "soft, moist, devitalized (nonviable) tissue. It may be white, yellow, tan, or green and it may be loose or firmly adherent"[11] (Fig. 8-2). Regardless of whether slough is or is not tissue, clinicians agree that it must be removed in acute and chronic wounds where the goal of care is healing.[4,8,9,11–13]

Figure 8-1. Eschar. In a wound that has become dehydrated, necrotic tissue turns thick, leathery, and black. This tissue is referred to as *eschar*. (Photo courtesy of Sharon Baranoski, RN.)

Figure 8-2. Slough. Necrotic tissue that's moist, stringy, and yellow (devitalized tissue) is referred to as *slough*. (Photo courtesy of Sharon Baranoski, RN.)

In general, removing necrotic tissue restores the local vascular supply to the wound and improves healing. However, debriding too much viable tissue can destroy the collagen structural framework for healing. Some wounds shouldn't be debrided at all. Exercise caution and consider conservative sharp debridement, for example, in dealing with necrotic ulcers in persons with compromised vascular supply or immunosuppression.[11] Debriding heel ulcers remains controversial because of problems with perfusion and the small amount of tissue

that covers the calcaneus bone.[8,11] The 2019 EPUAP NPIAP PPPIA clinical guideline states “Stable heel eschars or eschars in the presence of untreated peripheral vascular disease should not be debrided. However, if there is high suspicion of infection, heel eschars should be debrided to reveal the base of heel pressure injury to enable comprehensive assessment and treatment of the heel pressure injury. Nonviable, necrotic, or infected tissue should be removed via appropriate methods of debridement.”[11]

There is clinical consensus that a vascular assessment needs to be performed to ascertain if there is an adequate vascular supply to heal prior to debridement of ulcers in the lower extremities.[1,8,11] Controversy revolves around whether to debride stable, adherent eschar with no exudate or wounds with no clinical signs of infection.[1,8,11] Some clinicians believe that although caution is indicated, all necrotic heels should be debrided.[9,12] The 2019 EPUAP/NPIAP/PPPIA pressure injury guideline endorses that stable, hard dry eschar should be assessed for signs of infection (erythema, tenderness, edema, purulence, fluctuance, crepitus, and/or malodor) and then recommend debridement in the presence of adequate blood supply.[11] Wounds become larger with debridement, and this expectation should be discussed with the patient/family prior to the procedure.

Pyoderma gangrenosum is a neutrophilic disorder that has a raised translucent border that may be associated with a rapidly expanding central ulcer. This condition may be idiopathic or associated with underlying inflammatory bowel disease, rheumatoid arthritis, or myeloproliferative disease (leukemia/lymphomas). When the raised peripheral border is active, pyoderma gangrenosum is one example of a wound that should not be debrided in the typical clinical setting.[2] This raised active border indicates an acute inflammatory reaction, and debridement would stimulate the infiltrate to spread even more through a process called *pathergy.*[2] However, successful clinical management, without pathergy, has been reported using a combination of complete surgical excision, followed by NPWT, HBOT, and skin grafting.[14] Septicemia is another condition that requires serious caution before initiating debridement. The wounds of patients

with septicemia should not be debrided unless the patient is receiving adequate systemic antibacterial coverage.[2]

Practice Point

Monitor stable necrotic heels for odor and signs of edema, erythema, tenderness, fluctuance from fluid under the eschar, purulence, crepitus, malodor, or drainage that may signal the need for debridement.[11]

Practice Point

It's often important to watch the line of demarcation between viable and necrotic tissue for signs of further tissue breakdown or softening eschar.

Chronic wound care begins with treating the cause and patient-centered concerns, including pain and activities of daily living. Assess individual patients to determine whether the wound is healable, maintenance, or nonhealable.

A healable wound has adequate vascular supply and the cause of the wound can be treated. To assess adequate blood supply,[1,4,11] a palpable pulse in the foot indicates an arterial pressure in excess of 80 mm Hg that is usually enough blood supply for healing to occur. This finding could equate to an ankle–brachial pressure index of 0.6 or higher. If there is a sufficient blood supply and the cause has been corrected (compression for venous ulcers and pressure offloading for

diabetic neurotrophic foot ulcers), the ulcer is healable. For pressure injuries, the source of tissue damage needs to be corrected, including local pressure, poor nutrition, shear, or decreased mobility. Active debridement can be coupled with dressings to facilitate moisture balance and bacterial reduction if local critical colonization/localized infection is present.

A maintenance wound is one that has a sufficient blood supply for healing but is unable to heal due to patient (will not wear compression bandages) or health delivery system factors (plantar pressure redistribution footwear for diabetic foot ulcers was too expensive/not covered by the healthcare system).[4] Debridement and local wound care should then be conservative for maintenance wounds. This often includes the removal of nonviable slough (conservative local debridement) but not to bleeding tissue as with an active debridement with removal of healthy tissue to create bleeding tissue (an acute wound in a chronic wound). A nonhealable or palliative wound does not have enough blood supply to heal and/or the cause cannot be corrected (e.g., negative protein balance with untreatable secondary cancer). Debridement for nonhealable and maintenance wounds should have conservative debridement and limited to easy to remove soft slough with a topical antimicrobial (povidone–iodine or chlorhexidine and its related derivative PHMB—polyhexamethylene biguanide) with moisture reduction to also reduce local bacteria.[4]

Although it is acknowledged that debridement is a critical first step in preparing the wound bed for healing, all of the recommendations on debridement from the international EPUAP/NPIAP/PPPIA 2019 clinical practice guideline are at the B2 level of knowledge[11] (Table 8-1).

Table 8-1 Debridement Recommendations: EPUAP/NPIAP/PPPIA 2019 Pressure Ulcer/Injuries Clinical Practice Guideline

12.4	Avoid disturbing stable, hard, dry eschar in ischemic limbs and heels, unless infection is suspected. (Strength of Evidence = B2; Strength of Recommendation ↑↑)
12.5	Debride the pressure injury of devitalized tissue and suspected or confirmed biofilm and perform maintenance debridement until the wound bed is free of devitalized tissue and covered with granulation tissue. (Strength of Evidence = B2; Strength of Recommendation = ↑↑)

From European Pressure Ulcer Advisory Panel, National Pressure Injury Advisory Panel and Pan Pacific Pressure Injury Alliance. In: E. Haesler, ed. *Prevention and Treatment of Pressure Ulcers/Injuries: Quick Reference Guide*. 2019, p. 241.Strength of Evidence (SOE) and Strength of Recommendation (SOR) can be found under Appendix Clinical Practice Guideline EPUAP/NPIAP/PPPIA 2019.

WBP is the management of a wound to accelerate endogenous healing or to facilitate the effectiveness of other therapeutic measures.[1,3,4,12] The original TIME model—**T**issue nonviable or deficient, **I**nfection or inflammation, **M**oisture imbalance, **E**pidermal margin nonadvancing or undermining[3]—has been reconceptualized to the **DIME**© model (Fig. 8-3).[1,4,12] As mentioned previously, you should first identify and treat the cause of the wound, address patient-centered concerns, and finally provide local wound care (Table 8-2).[4,12]

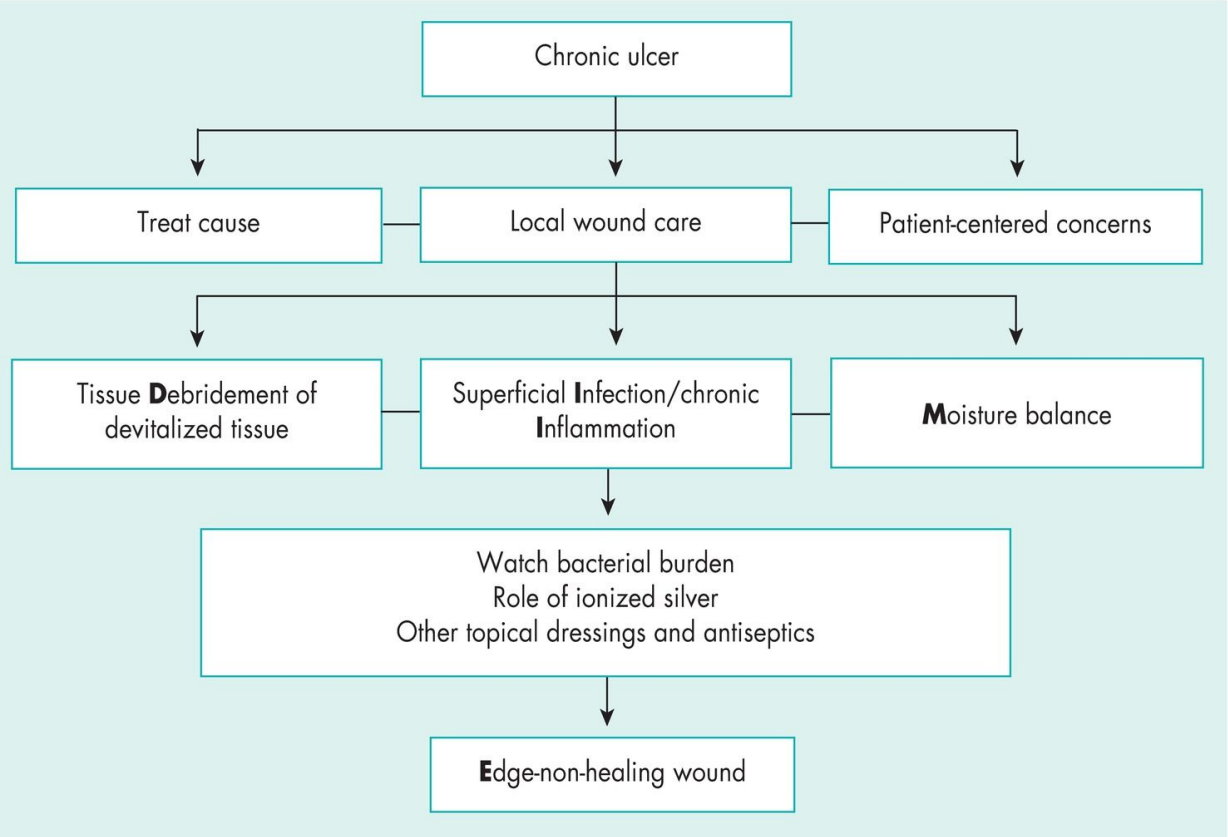

Figure 8-3. TIME is now DIME: Wound bed preparation DIME model.

Table 8-2 TIME is Now DIME: Wound Bed Preparation

	Chronic ulcer	
Treat cause	Local wound care	Patient-centered concerns
Tissue **D**ebridement of devitalized tissue	Superficial **I**nfection/chronic **I**nflammation	**M**oisture balance
	Watch bacterial burden Role of ionized silver Other topical dressings and antiseptics	
	Edge—nonhealing wound	

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Practice Point

Save time and use the DIME[1,3,4,12] acronym in preparing the wound bed for healing (healable wound) or optimizing local wound care in maintenance or nonhealable wounds. Local wound care includes:

- **D**ebridement
- **I**nfection or inflammation
- **M**oisture imbalance
- **E**dge—nonhealing

Predebridement Teaching

Patient-centered care should include teaching about the purpose and usual expectations of the debriding process. Before initiating as a treatment, the process needs to be explained and understood by the patient, their family, and their circle of care. Circle of care is an expression, which includes the individuals and activities related to the care and treatment of the patient. Thus, it covers the healthcare providers who deliver care and services for the primary benefit of the patient. Include in your teaching the debridement options that could be used and the method chosen should be acceptable to the patient and clinician with the desired outcome and potential complications. It is vital that the patient and family understand why the necrotic tissue is being removed. The importance of educating patients and their families and obtaining written consent prior to debridement is one of the ten recommendations for conservative sharp wound debridement.[15] Some laypersons mistakenly believe that necrotic tissue is a “scab” (eschar) and is a sign of healing. Epithelium needs a firm pink surface of granulation tissue base at the wound edge to migrate optimally toward the center of a wound. Delayed healing will result if epithelial margins need to migrate down valleys under eschar or over hypertrophic or unhealthy bright red friable granulation tissue. Similarly, patients and families need to know that the wound will change during the debridement process because an acute wound is created within a chronic wound to stimulate the healing process. For example, tell them to expect that the wound may become larger in size.

Another patient-centered concern within the WBP model is pain. (See chapter 12, Pain Management and Wounds for an extensive discussion on pain.) Adequate wound debridement needs to consider how debridement pain will be managed. In an outpatient setting, for example, a wound clinic or practitioner’s office, application of a topical local anesthetic agent prior to debridement may be adequate for small-size wounds. For larger wounds requiring more extensive debridement, surgical debridement may need to take place in the operating room where an anesthetic practitioner (anesthesiologist,

family practitioner, or nurse with anesthetic training) can administer regional or general anesthesia. As with any surgery, a preoperative evaluation of the patient is performed by the anesthesiology practitioner to assess individual patient needs.[16]

Wound care patients may have more comorbidities that need to be identified and evaluated prior to debridement. In one medical center, there were higher operative risks identified through higher American Society of Anesthesiologists (ASA) scores for wound patients (3.09) compared to other surgical patients (2.09).[16] Another safety example is a patient with a cardiac pacemaker or internal defibrillator will need a comprehensive assessment including knowing the specific type and manufacturer of any implanted devices and assessing the cardiac device precautions or protocol *before* the patient arrives on the operating table.[17] Some pacemakers (mostly older models) may malfunction with electrocautery devices used for hemostasis of bleeders.

If a patient is unable to express their pain experience, a family member may be advocating for them so that they do not experience excessive pain.[18] One such patient population includes patients with the locked-in syndrome (LIS). LIS can occur from a basilar artery thrombosis that results in a ventral pontine infarction leaving the patient without motor function. Although the patient cannot move or speak, and even though the patient's eyes may be closed, the patient may be awake and conscious and thus able to experience pain.[18] Special monitoring in the OR using bispectral (BIS) monitoring will facilitate adequate pain management during debridement.[18] Patients, their family, and circle of care need to understand that either regional or general anesthesia may be appropriate choices for debridement pain management in the OR.[19]

Debridement Methods

Mechanical, sharp/surgical, enzymatic, and autolytic are the common methods of debridement.[1,2,4,8,11] However, a resurgence in the use of older methods including maggots (biological or larval therapy) has become accepted practice in some wound care centers (Table 8-3).[2,4]

Table 8-3 Best Practices for Preparing the Wound Bed

Identify and Treat the Cause
• Diagnose and correct or modify treatable causes of tissue damage. • Differentiate the wound's ability to heal: healable, maintenance, or nonhealable wound.
Address Patient-Centered Concerns
• Assess and support the management of patient-centered concerns to enable healing. • Provide patient education and support to increase adherence to the treatment plan.
Provide Local Wound Care
• Assess and monitor the wound history and physical characteristics (location and measurements). • Debride healable wounds with adequate vascular supply, removing nonviable, contaminated, or infected tissue (surgical, autolytic, enzymatic, mechanical, or maggot). • Clean wounds with low-toxicity solutions (such as normal saline or potable water); reserve topical antiseptic solutions for wounds that are nonhealable or those in which the bacterial burden is of greater concern than the stimulation of healing. • Assess and treat the wound for increased bacterial burden or infection; distinguish from persistent inflammation of nonbacterial origin. • Select a dressing that is appropriate for the needs of the wound, the patient, and the caregiver or clinical setting. • Monitor the quantity and quality of wound exudate to prevent periwound maceration. • Evaluate expected rate of wound healing; if suboptimal, reassess patient.
Provide Organizational Support
• For improved outcomes, education and evidence base must be tied to interprofessional teams with cooperation of healthcare systems.

Reprinted with permission *Wound Care Canada*, the official publication of the Canadian Association of Wound Care. Available at: http://www.cawc.net, © 2006. All Rights Reserved; Sibbald, R.G., et al. "Best Practice Recommendations for Preparing the Wound Bed: Update 2006." *Wound Care Canada* 4(1):15-29, 2006.

Mechanical Debridement

Methods of mechanical debridement include wet-to-dry dressings, hydrotherapy (whirlpool), and wound irrigation (pulsed lavage).[1,3,4] Mechanical debridement may be more painful than other debridement methods, and the healthcare provider should always consider patient premedication for pain. Both whirlpool and pulsed lavage require special equipment and skill. The spread of bacteria and possible infection are also potential complications of mechanical methods especially whirlpool and pulsed lavage methods.

All of the mechanical methods are considered nonselective debridement; that is, they don't always discriminate between viable and nonviable tissue. Mechanical methods may be harmful to healthy granulation tissue on the surface of the wound and lead to bleeding, trauma, and disruption of the collagen matrix along with the necrotic tissue.

Practice Point

Mechanical debridement may be painful; consider patient premedication for pain and the availability of other debridement options.

Wet-to-Dry Dressings

Despite the drawbacks, such as pain and the necessary application up to three times per day, the use of wet-to-dry wound debridement dressings unfortunately remains a common treatment in some healthcare settings. This method involves placing a moist saline gauze dressing on the wound surface and removing it when it's dry. The removal of the dried gauze dressing facilitates removal of devitalized tissue and debris from the wound bed. However, newly

formed granulation tissue and new cell growth are also removed. To prevent pain and to help remove the dry gauze, clinicians often wet the dressing before removal. However, this defeats the purpose of aggressively removing dead tissue. This type of debridement requires significant nursing time, and although the materials may be relatively inexpensive, the overall cost including the need for pain medication can often be greater than other techniques.

A wet-to-dry dressing can be used when a moderate to large amount of necrotic tissue is present and surgical intervention is not an immediate option. Because of pain and the removal of viable tissue, the Centers for Medicare and Medicaid Services (CMS) has stated in its revised surveyors guidance on pressure injuries in long-term care that use of wet-to-dry dressings should be limited.[20] CMS also states that wet-to-dry dressings should not be used in a clean, granulating wound. Instead, they recommend use of moist wound therapy dressings.

Pulsed Lavage

Pulsed lavage debridement may be indicated for patients with large amounts of necrotic tissue and for those in whom other debridement methods are not an option, thus it is most commonly employed in the operating room. It is accomplished by using specialized equipment that combines a pulsating irrigation fluid with suction.[21] Utilizing pulsed lavage, you can clean and debride a wound at variable irrigation pressures (measured in pounds per square inch [psi]). The pulsatile action and effective wound bed debridement may improve granulation tissue growth. This treatment takes 15 to 30 minutes and should be performed twice daily if more than half of the wound contains necrotic tissue.

Patients typically require premedication for comfort before beginning the procedure. Safe and effective ulcer irrigation pressures range from 4 to 15 psi.[22] Water pressure control is critical during this procedure. Because fluid is being forced directly at the wound, the risk of driving organisms deep into the wound tissue is a concern. In addition, inhalation of contaminated water droplets or mist is possible for both the clinician and the patient.

Practice Point

Always use appropriate equipment to prevent excess lavage pressure. Wear personal protective equipment to prevent splash injury, including eye and face protectors as well as an impervious gown. Remember to administer pain medication to the patient before the procedure. Based on the logistics and equipment, concern for infection control–related issues such a splash, splatter and aerosolization, and requirement for pain control, this technique is rarely utilized in the wound care clinical setting.

Sharp/Surgical Debridement

Sharp/surgical debridement includes the use of a scalpel, curette, forceps, scissors, ultrasonic or hydrosurgery devices, or lasers to remove dead or devitalized tissue.[2,4,23] Sharp debridement is considered by many clinicians to be the gold standard of debridement.[24] It can cause pain, so a topical anesthetic including lidocaine cream or gels are often used and premedication with systemic pain medication may be required.[25] Patients may also need follow-up appointments for serial debridement.

Because viable tissue may also be removed inadvertently with this method, excellent judgment must be used when performing sharp debridement.[24–26] Training and experience are mandatory as the clinician must be able to differentiate where and what to cut, identifying, for example, a tendon versus slough because both are yellow in color[24–27] (Fig. 8-4). Skill is required as clinicians must be able to discern the line of demarcation between viable and nonviable keratinocytes at the wound edge. As shown in Figure 8-5, most clinicians can identify the stalled edge of a chronic wound (location A), where the surface keratinocytes, at the wound edge, cannot migrate across the wound bed. Research suggests that the wound edge really extends to location B[28,29] and that debriding only to this boundary may be inadequate as it leaves behind impaired cells.[28,29] Therefore, adequate debridement of a chronic wound needs to extend beyond the point at which the keratinocytes have lost the ability to move (edge of the callus tissue at location B) to where the cells can move to heal the wound (location C). Clinicians may need to rethink the distance from the wound edge (location A) that they need to debride. Research focuses on an easy way for clinicians to identify how far from the wound edge to perform debridement, based on the pathology of the abnormal keratinocytes at the wound edge.[28,29] While this technique is being adapted for clinical use, Endara and Attinger have described a simple clinical technique to stain the tissues in vivo with methylene blue dye as a color guide to aid the clinician to do a complete wound debridement[27] (Fig. 8-5).

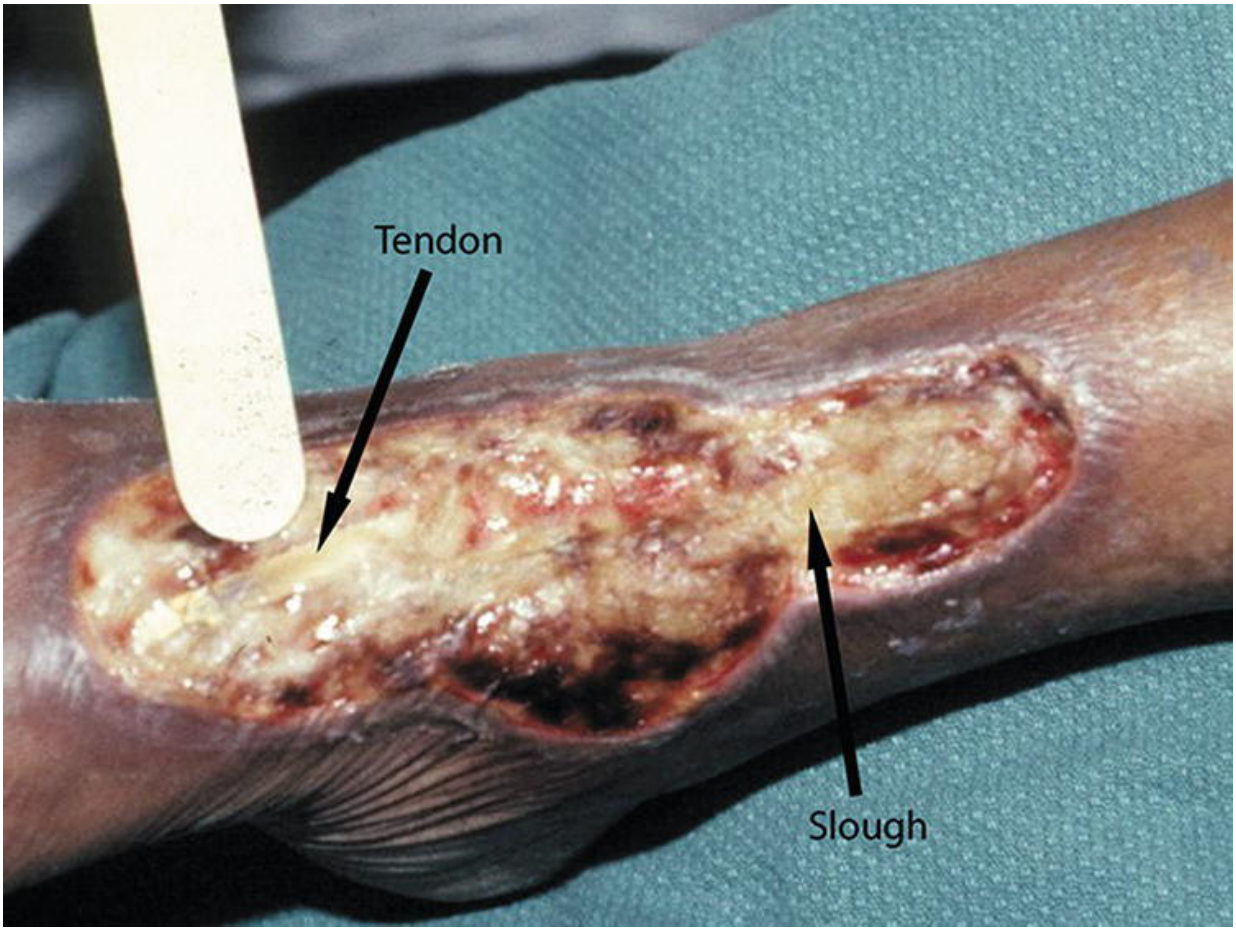

Figure 8-4. Differentiating tendon from slough. Performing debridement requires knowing where and what to cut. For example, tendon and slough both are *yellow*—the clinician must be able to distinguish between them. (Photo courtesy of Sharon Baranoski, RN.)

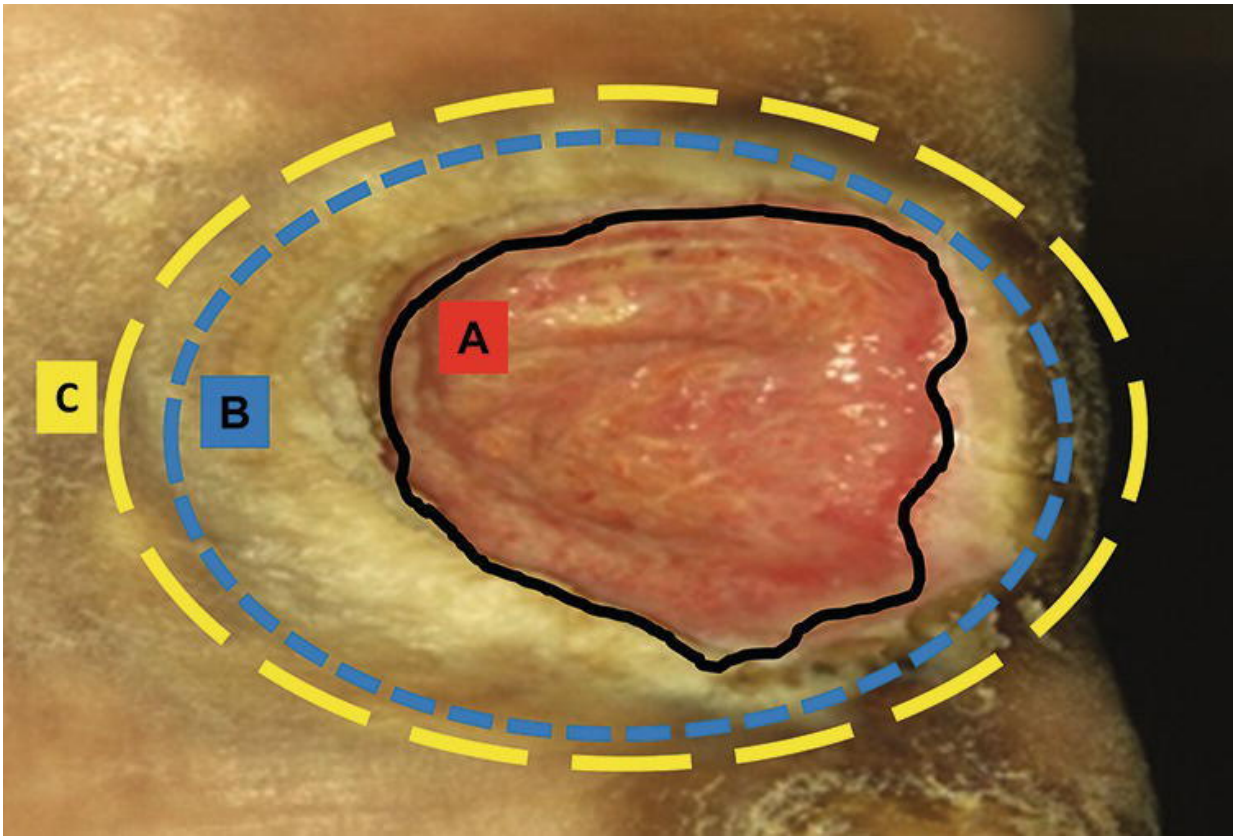

Figure 8-5. Margin of debridement. The *solid black line* **(A)** indicates a nonhealing edge. The *outer blue and yellow broken lines* **(B and C)** indicate two possible margins of debridement. Location B is the edge of the wound, whereas location C is the presumed location of the healing edge of the wound where keratinocytes have the ability to migrate and participate in the wound healing process. Therefore, debride to location C. (Adapted from Tomic-Canic, M., Ayello, E.A., Stojadinovic, O., et al. "Using Gene Transcription Patterns [Bar Coding Scans] to Guide Wound Debridement and Healing." *Advances in Skin & Wound Care* 21(10):487-92, 2008.)

Conservative (selective) sharp debridement that removes nonviable tissue from the wound base with minimal bleeding must be distinguished from surgical (excisional) sharp debridement that transforms a chronic wound into an acute wound by completely excising all nonviable tissue and exposes healthy, bleeding, viable tissue.[1–3,12,24] Surgical excisional debridement is usually performed by experienced physicians or surgeons. In the United States, only physicians can perform surgical debridement. This can be accomplished on a limited basis in the wound care center, but more commonly is performed in the controlled setting of the operating room or surgical center. Individual states may allow nurses, physical therapists, and physician assistants with appropriate licensing and training to perform some sharp debridement procedures with a scalpel, forceps, or scissors. Recommendation for conservative sharp debridement for nurses in the United States is written by Wound Ostomy & Continence Nursing Society®[30] and for Canada.[15]

The use of sharp debridement is based on expert opinion and clinical data. Steed et al.[31] reanalyzed data from a six-site random-controlled trial that tested the use of recombinant human platelet–derived growth factor (rhPDGF) versus placebo in neuropathic diabetic foot ulcers. These authors found significantly higher healing rates in treatment facilities in which more frequent and complete surgical debridement to bleeding tissue was performed rather than simply removal of the periwound callus. The removal of loose bright friable granulation tissue from the surface of an ulcer removes senescent fibroblasts as well as bacteria that may be arranged in biofilms. The wound surface bacteria and callus around the perimeter of the ulcer lead to underlying tissue damage.

Sharp (selective) debridement is used for adherent eschar and devitalized or dead slough on the wound surface. Surgical (excisional) debridement is often selected for acute infected wounds and should be the first choice for wounds with extensive tissue necrosis or those demonstrating signs of advancing cellulitis or sepsis. Small wounds may be debrided in the wound care clinic or at the bedside, but extensive wounds—for example, a stage 4 pressure injuries—may require debridement in the operating room. A variety of instruments can be utilized for sharp débridement including curettes, iris scissors

and pickups, scalpels, and ultrasonic devices. Of these débridement tools, only the ultrasonic devices have the ability to debride as well as deliver energy to the wound (acoustic steaming and cavitation) that result in bacterial killing as cellular stimulation.[32] Physicians have reported that the use of hydrosurgery devices has decreased the number of times that debridement was needed[23] (Fig. 8-6). Other authors have described the use of hydrosurgery devices in combination with undiluted Betadine and a bilayer porcine collagen dressing to successfully debride burns.[33] Surgical/sharp debridement must be performed with extreme caution in patients taking anticoagulant medications. The medication may need to be held for a short period of time prior to the procedure. Patients with prolonged bleeding may be best treated with other methods of debridement (Figs. 8-7 and 8-8).

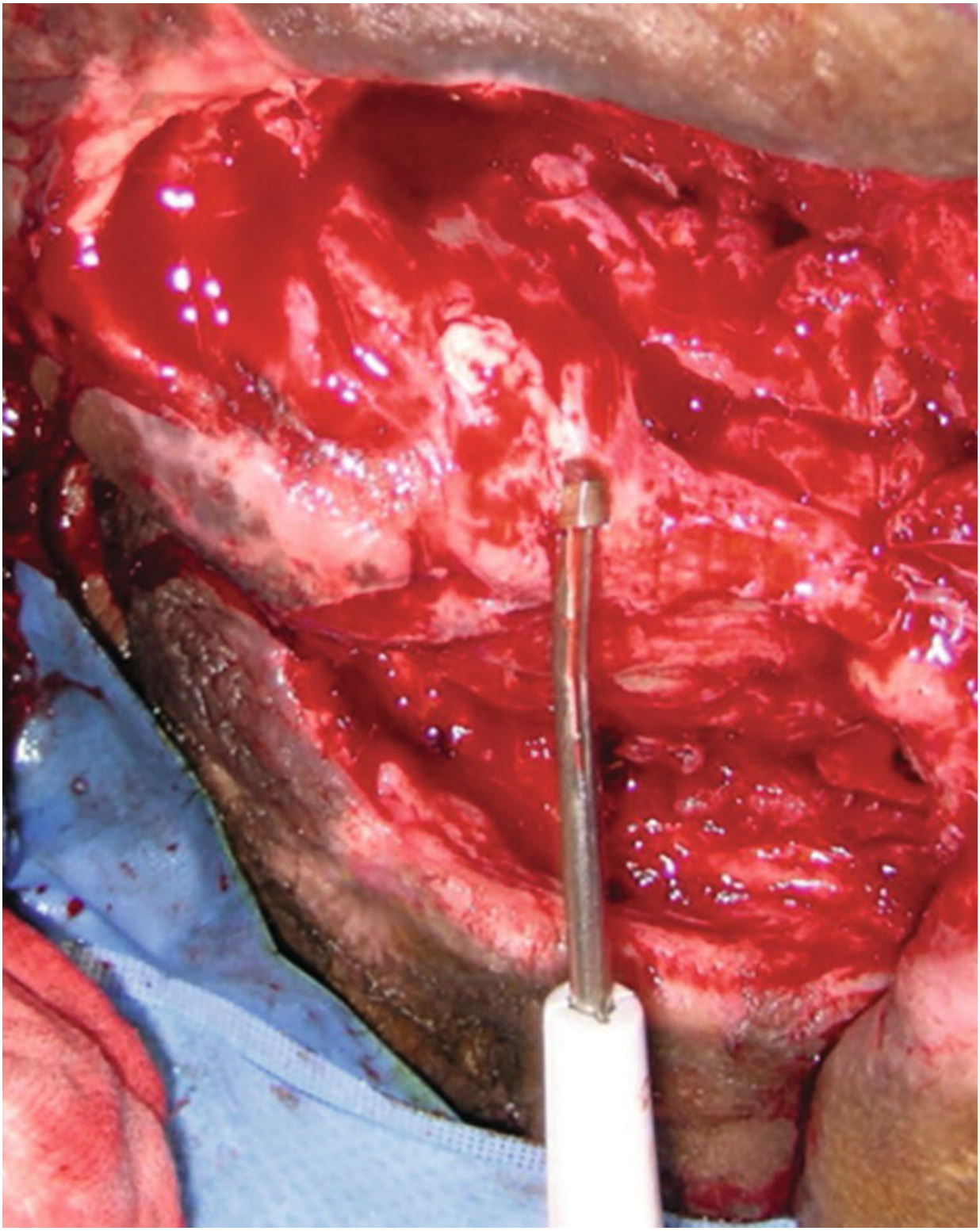

Figure 8-6. Hydrosurgery device. Example of one type of device used to remove necrotic tissue. (Photo

courtesy of Jeffrey Niezgoda, MD.)

Figure 8-7. Sharp debridement using a curette. (Photo courtesy of Jeffrey Niezgoda, MD.)

Figure 8-8. Sharp debridement using iris scissors and pickups. (Photo courtesy of Jeffrey Niezgoda, MD.)

Sharp debridement can also disrupt biofilm. Wolcott used both sharp debridement and antibiotic treatment to demonstrate a nine-fold reduction in bacterial concentration compared to patients without debridement and only antibiotics were used.[34] However, the impact of sharp debridement alone against biofilm is short-lived. Kim reported that biofilm can reform as quickly as 6 hours following a débridement procedure.[35]

Post active sharp debridement, calcium alginate dressings (rope or wafer) can be placed on the acute wound. The calcium alginate donates calcium and will facilitate hemostasis with mild oozing and external pressure on the dressing. The alginate accepts sodium that creates a hydrogel preventing the formation of a hemorrhagic crust.

This allows the wound to move from the inflammatory to the proliferative stage without the inhibition of a surface proinflammatory hemorrhagic crust.

Practice Point

Exercise caution when performing surgical/sharp debridement on any patient who has been on a prolonged course of anticoagulant therapy.

Enzymatic Debridement

Enzymatic debridement is considered safe, effective, and easy to perform. Enzymes are effective wound surface cleaning agents that accelerate eschar degradation and debridement. The removal of debris helps a chronic wound move from the inflammatory stage to the proliferative stage, resulting in enhanced wound healing.[36] Enzymatic agents are an ideal option for patients:

- who are not candidates for surgery
- in combination with surgical debridement of slough to prevent secondary infection
- receiving care in a long-term facility or at home where there is often a lack of qualified staff for surgical debridement
- requiring serial debridement.[37]

Enzymatic debridement is accomplished by applying topical enzymatic agents daily to devitalized tissue. These agents will digest and dissolve necrotic tissue in the wound bed by breaking down collagen, elastin, and other components of the abnormal devitalized wound matrix.[2,4,12] Before applying the topical enzymatic agent, crosshatching or scoring the eschar without causing bleeding (if allowed by state licensing laws) can be performed to enhance local penetration of the agent[38] (Fig. 8-9).

Figure 8-9. Application of enzymatic debridement agents. (Photo courtesy of Jeffrey Niezgoda, MD.)

If infection has spread beyond the ulcer (as in advancing cellulitis), immediate removal of necrotic tissue is usually recommended, ideally in conjunction with systemic antimicrobials. Surgical debridement and then possible enzymatic debridement should be considered. Enzymes often can be used alone, to break down the eschar before or after sharp debridement, or in conjunction with mechanical debridement.[37] The slough produced from enzymatic debridement often facilitates bacterial growth and makes some patients susceptible to deep and surrounding infection. Some topical antiseptic nonrelease dressings (polyhexamethylene biguanide [PHMB] gauze or foam, methylene blue–crystal violet foam) are compatible with enzymatic debriding agents and may be used in conjunction with the treatment.[39] (For more information on dressing treatments, see Chapter 9.)

Enzymes that act on necrotic tissue are categorized as proteolytics, fibrinolytics, and collagenases, depending on the tissue component they target. Because papain–urea enzymatic debriding agents target eschar, they are often used on necrotic wounds. However, the Food and Drug Administration removed papain–ureas from use in the United States as of 2008.[40] Those practicing outside the United States, where papain–urea is available, are urged to review the literature about this enzymatic debriding agent.[36,38,41–43] Collagenases target nonviable collagen tissue while sparing viable tissue, thereby making them useful in necrotic wounds with slough tissue at the wound base.[39]

Practice Point

Silver and other metals, including zinc oxide, can inactivate enzymes; be careful when selecting cleaning agents and dressings that contain any ingredients that can interfere with enzymatic action.[39]

Enzymes are a prescription drug and require a physician's or prescriber's order. Since they are drugs, they must be signed for by the nurses in the patient's medication record. Collagenase is usually applied once per day. Before reapplying any enzymatic agent, thoroughly clean the wound with normal saline or a wound cleanser to remove any residual enzymatic ointment and loose wound debris.[38] Avoid solutions or dressings with metal ions including mercury or silver that may inactivate the enzymatic action of collagenase.[39]

If it is within your state's scope of practice and permitted by your facility, crosshatching or scoring the eschar with a scalpel (No. 11 or No. 15 blade), without cutting deep enough to cause bleeding, is recommended prior to applying the enzyme to let the debriding agent penetrate into the eschar. After scoring is complete, apply a thin layer (about the thickness of a nickel) of enzymatic ointment onto the necrotic tissue. Cover the wound with an appropriate dressing to keep it moist and let the debriding agent work.[38,39,44]

Collagenase is derived from *Clostridium histolyticum*. It may be more effective than papain–urea at degrading collagen and elastin and is thought to work from the bottom of the wound up.[42] Research supports collagenase as a more selective enzyme that may be more effective in controlling or reducing pain when compared with other debridement methods.[38,45] Collagenase up-regulates the migration of keratinocytes over the wound bed and stimulates granulation.[8,46,47] Muller et al. documented debridement with collagenase to be quicker and more cost-effective than autolytic debridement with a hydrocolloid dressing in 24 patients with pressure ulcers.[48] Research by Riley and Herman[49] has shed new light on the role keratinocytes play in wound healing and how wound healing is influenced by different substances, including enzymes. This research assessed that collagenase doubled keratinocyte growth and migration; the increase was further enhanced

fivefold when heparin-binding epidermal-like growth factor was added.[49] Mekkes et al. in vitro study detected increased healing rates with collagenase and ineffective debridement with fibrinolysin (deoxyribonuclease).[50] Collagenase has also been shown to reduce scarring in partial-thickness burn wounds that has crucial implications for a patient's quality of life.[51]

The optimal method for enzymatic debriding agents has been the subject of much controversy. Research has also elucidated some differences between papain–urea and collagenase. While overall healing rates for papain–urea and collagenase were not significantly different in one study, papain–urea did debride eschar at a faster rate.[52] However, a second study found that papain–urea decreased keratinocyte migration by 50%.[49]

The search for new enzymes continues. Mekkes et al. have reported on Antarctic krill as an effective debriding agent,[53] while a systematic review by Bradley et al.[54] failed to provide evidence to support the use of enzymatic agents over other methods such as hydrogels or hydrocolloids for autolytic debridement.

Autolytic Debridement

Autolytic debridement uses the body's endogenous enzymes to slowly remove necrotic tissue from the wound bed. In a moist wound, phagocytic cells and proteolytic enzymatic enzymes can soften and liquefy the necrotic tissue that is then digested by macrophages. Autolytic debridement can be facilitated with appropriate dressings in the superficial wound that contains little necrotic tissue or a larger, deeper pressure injury.[1,2,4,12] Underlying these concepts is the requirement of adequate circulation and nutrition.[2] Autolytic debridement may take longer than some other methods; however, it represents a less stressful method to the patient and wound than mechanical debridement. Autolytic debridement is contraindicated in deep and surrounding infected wounds.

Autolytic debridement is easy to perform and involves applying a semiocclusive or occlusive moisture-retentive topical dressing including transparent films, hydrocolloids, hydrogels, and calcium alginate dressings.[2] Wound fluid accumulates under the dressing, aiding in the lysis of necrotic tissue. This method is relatively pain free in patients with adequate tissue perfusion.

Studies have compared the efficacy of hydrogel dressings and mechanical debridement with wet-to-dry dressings.[55–57] Several researchers[54–57] have concluded that autolytic debridement with a hydrogel is more time-efficient and cost-effective, resulting in faster healing when compared with wet-to-dry dressings. One case report found that use of a clear acrylic dressing promoted autolytic debridement and had the added advantage of being able to view the wound without removing the dressing.[58] Schimmelpfenning and Mollenhauer[58] as well as Konig et al.[59] found that autolytic debridement with moist interactive dressings was equal in efficacy to enzymatic methods using collagenase. Smith's *Cochrane* review on diabetic foot ulcer debridement concluded that hydrogels were more effective than gauze.[60] A case report by Cuschieri et al. reported the successful autolytic debridement of a large necrotic foot ulcer in a diabetic patient.[61]

Practice Point

Be sure to tell the patient and family that fluid accumulating under the dressing is a normal part of the debridement process. Discolored wound fluid may not signal a wound infection.

Monitor the wound for signs of infection including odor, increasing exudate or increasing wound size, periwound erythema and/or edema, warmth, or increased pain. Discontinue autolytic debridement if these symptoms occur. Immunocompromised patients should be assessed frequently for any clinical indication of infection. Autolytic debridement isn’t the treatment of choice in deep and surrounding wound infections; in fact, it may lead to more severe infection and is therefore contraindicated in these situations. Surgical consult is warranted with appropriate medical management of the infection.

Maggot Therapy (Biological or Larval Therapy)

Maggot therapy was widely used in the early part of the 20th century. It fell out of favor due in part to the “disgust factor” and the use of newer modalities such as antimicrobial agents in wound treatments. With Europe leading the way, there has been a resurgence in the use of larval therapy in the United States.[62,63] In this type of debridement, a single or several applications of sterilized medicinal *Lucilia sericata* (green bottle fly) maggots are placed in the wound bed every 2 to 3 days[64] (Fig. 8-10). The specific application technique for how the maggots are actually put in the wound varies. Some place the maggots directly into the wound so they can roam around (free range), and others place the maggots contained in a device such as a pouch or tea bag–like sack.[65] Early scientific reports of the evidence support that free-range maggot therapy maximizes debridement benefits compared with maggots that are contained in pouches or sacks and then placed in the wound.[65]

A

B

C

Figure 8-10. Maggots in heel wound. **(A)** This photo shows a heel ulcer with osteomyelitis on a middle-aged woman with diabetes who was on immunosuppressants following a kidney transplant 27 years earlier. **(B)** The same ulcer with sterile maggots placed in the wound for the purpose of debridement. **(C)** The same ulcer after removal of the first application of maggots. **(D)** The same ulcer, healed.

(Photos courtesy of Pamela Mitchell, BTER Foundation.)

Just how do these larvae accomplish debridement? The mechanism by which maggot therapy works is believed to be by the enzymes the maggots secrete. These substances are proteinases that degrade the necrotic tissue.[66] The maggots also digest bacteria, making them effective in wounds with resistant bacteria strains.[67,68] A prospective randomized study provided evidence that complete lysis of methicillin-resistant *Staphylococcus aureus* (MRSA) and *Candida albicans* occurred within 24 hours when maggots were applied to 48 culture plates.[69] This effect continued to be observed for 5 days.[69] Maggots also encourage healing by stimulating granulation tissue.

Prospective trials on the efficacy of maggot therapy have reported debridement and evidence of decrease in wound size.[1,70,71] Some case studies have reported the efficacy and selectivity of maggot debridement therapy.[72,73] Mumcuoglu[74] has reported significant debridement (80% to 85%) with clinical application of maggots as well as the prevention of amputation and bacterial spread.

Maggots can be used in almost all wounds. Richardson[75] and Thomas[76] reviewed current best practices. However, some contraindications for their use include a life- or limb-threatening wound, psychological distress or the “ick factor,” bleeding abnormalities, and deep-tracking wounds.[62] The literature is unclear about some aspects of maggot use. Sherman[77] warned clinicians that maggots should not be used in the presence of osteomyelitis or critical ischemia associated with arterial insufficiency, but Claxton et al. disagree.[62] Maggots are cost-effective when used properly and have the potential to help in situations where other resources are not available.[78] Despite the potential for psychological distress, most patients, as well as healthcare professionals, were satisfied with the treatment in one study.[72]

Something else to consider with maggot therapy is the level of pain it causes. There are conflicting reports about the level of

expected pain with maggot debridement therapy. Sometimes, pain is minimal or absent.[79] In one report, however, 25% of patients with superficial painful wounds complained of increased pain during treatment with maggots.[66] These patients need analgesics for pain management. The type of wound might make a difference as to whether the patient has pain during therapy. In a retrospective study, Steenvoorde et al.[80] determined that diabetic patients, the majority of whom had neuropathy (*n* = 21), did not experience increased pain, while 40% (*n* = 8) of the enrolled nondiabetic patients experienced pain when being treated with maggots.

Choosing a Debridement Method

Does it matter which type of debridement you use on a wound? “No one method of debridement has been proven optimal for pressure injuries,” according to the Wound, Ostomy and Continence Nurses Society (WOCN).[8]

Given the various and conflicting evidence about the different debridement options, choosing the right way to debride wounds can be challenging. Answering the following questions can help guide you in choosing the best debridement method for your patient.[1,2,4,11,12,15,81] Remember, your choice may be limited by the availability of the various debridement methods in your facility or healthcare system.

• Determine the Ability of the Wound to Heal

Is there enough blood supply to heal?

Check that the Cause Has Been Corrected?

If you cannot correct the cause or there is inadequate blood supply to heal, conservative debridement of slough, moisture reduction, and bacterial reduction are achieved by conservative surgical removal of the slough and bacterial/moisture reduction through antiseptics such as povidone–iodine or chlorhexidine/PHMB preparations.

• What Are the Wound Characteristics?

Consider the size, depth, location, amount of drainage (and whether it is increasing), presence (and extent) or absence of infection, and etiology of the wound.

• How Much Time Do You Have to Debride?

Infected wounds require immediate attention and may require surgical debridement after systemic antimicrobial therapy has been initiated. The patient’s clinical condition and the amount of time that you can devote to a treatment may influence your choice.

• What Methods Are Permitted?

Check that the intended debridement method is allowed by your state's practice act and by your facility. For example, using a scalpel to crosshatch eschar requires specialized training and licensure.

• How Selective a Method Is Needed?

Determine the risk for damage to healthy tissue when necrotic tissue is removed.

• What's the Care Setting?

Some resources available in a hospital aren't practical in the home and may not even be available in a long-term care facility.

• How Much Debridement Is Enough?

How do you know when you've debrided enough? Assess the tissue in the wound bed: When most of the wound surface is covered with granulation tissue and the necrotic tissue is gone, you've debrided enough. As previously mentioned, Endara and Attinger describe their clinical technique to stain tissues with methylene blue dye prior to wound debridement so that the clinician can readily see the end point for surgical debridement.[27]

There are some methods supported by research that can guide practice for evaluating the effectiveness of debridement. Saap and Falanga[82] developed a method to assess adequacy of wound debridement. Their Debridement Performance Index may make more effective comparisons between different debridement methods and facilitate more predictive prognostic information.[82]

Falanga[83] has proposed that chronic wounds need constant debridement because serial debridement is an important part of the WBP in these wounds. In the past, debridement was regarded as a singular event based on the visible assessment of the wound. Now, however, it's thought that frequent, limited, serial debridement may keep the biological burden low and stimulate growth factors[12,84–86] (Fig. 8-11). Maintenance debridement may prove challenging in the

future due to proposed reimbursement changes in the United States based on the place in which debridement can be performed as well as the frequency of the procedure.

Figure 8-11. Serial photographs demonstrating effectiveness of selective sharp debridement. (Photo courtesy of Jeff Niezgoda, MD)

Summary

Debridement is an essential part of the WBP management process. Although surgical debridement is the fastest way of removing necrotic tissue from a wound, it may not be appropriate for all patients in all healthcare settings. The selection of the correct method of debridement should be based on the individual patient and the degree of necrosis present. By knowing the options for debridement, you can help prepare the wound bed and assist your patient on the road to healing. Remember to address patient-centered concerns including pain prior to proceeding with the selected debridement method (Table 8-4).

Table 8-4 Overview of Debridement Methods

Method	Considerations	Contraindications
Surgical/Sharp		
Necrotic tissue is removed using a scalpel, scissors, forceps, or curette.	• Urgent need for debridement • Highly selective • Rapid results • Pain unless the patient has neuropathy; analgesia often needed • Risk of hemorrhage/complications • Cost; use of special equipment • Requires patient consent • Requires special training and expert confidence level (including anatomic knowledge) • Must distinguish between necrotic and healthy tissues • Can be done at bedside • May require an operating room and systemic anesthetics for extensive procedures • Anticoagulant therapy may need to be held prior to the procedure	• Malignant wounds • Patients with clotting/ bleeding abnormalities or on anticoagulants • Ischemic tissue • Unstable medically • Underlying dialysis fistula, prosthesis, or arterial bypass graft • Caution with wounds involving hands and face • Caution with immunocompromised patients

Method	Considerations	Contraindications
Autolytic		
Endogenous enzymes present in wound fluid interact with moist dressing to soften and remove necrotic tissue.	• Need for minor or moderate debridement • Patient has a decreased or minimal risk of wound infection • Performed in any setting • Can be used with other methods • Selective • Safe, easy to use • Relatively painless and soothing when dressing in place • Slow • Risk of maceration to surrounding skin • Removal of some dressings may be painful • Odor • Secondary dressing needed for some types of primary dressings • Absorptive dressing can dehydrate the wound bed	• Some dressings cannot be used with infected wounds • Exposed tendon/bone • Friable skin • Deep extensive wounds • Severe neutropenia • Immunocompromised patients
Mechanical		
Wet-to-dry: Moist dressing is applied to wound, allowed to dry, and removed with force.	• Larger wounds • Nonsurgical candidates • Nonselective • Painful • Frequent dressing changes required, may need to be done 2–3 times a day; not cost-effective • May macerate surrounding skin • Bleeding • Dressing fibers stick to wound and can cause a foreign body reaction • May disperse bacteria into the air when removed • Traditional more than a modern accepted practice	• Clean wounds
Hydrotherapy: Moving water dislodges loose debris.	• Increases circulation to wound bed • May macerate periwound skin • Time-consuming • May cause trauma to wound bed and lead to bacterial contamination of wound and environment	• Clean wounds • Presence of diabetic neuropathy
	• Labor-intensive • Theoretical risk of fluid embolism or promotion of infection with irrigation • Healthcare professional needs personal protective equipment due to aerosolization • Can impede venous blood flow in legs	

Method	Considerations	Contraindications
Pulsed lavage: irrigation combined with suction	• Bedbound patients • Wound with large amount of necrotic tissue	• Clean wounds
Maggot Larvae (*Lucilia sericata*—Green Bottle Fly)		
Break down necrotic tissue and digest bacteria	• Psychological distress to patients or clinicians • Allergic reaction • Potential for increased pain especially in ischemic wounds • Time-consuming • Selective debridement • Rapid • Costly • May be painful—except with neuropathy • Decrease bacterial load • Bedside use • Can be used for various wound types, including infected wounds	• Allergies to adhesives, fly larvae, eggs, soybeans • Patients with bleeding abnormalities • Deep, tunneled wounds
Enzymatic		
Enzymes degrade and remove necrotic tissue.	• Patient on anticoagulants • Can be used on infected wounds • Cost-effective • Bedside use • Selective debridement • Decreased wound trauma • Cost varies • Usually daily application in United States • Sting with application/ inflammation around wound with some enzymes • Not used with heavy metal salts (silver and mercury) • May need crosshatching of eschar • Clinicians need to document in patient's medication record because enzymes are prescribed drugs	• Clean wounds • Allergy to component of the enzyme preparation

From Kirshen, C., et al. "Debridement: A Vital Component of Wound Bed Preparation." *Advances in Skin & Wound Care*, 19(9):506-17, quiz 518-9, November-December, 2006; Sibbald, R.G., Goodman, L., Woo, K.Y., et al. "Special Considerations in Wound Bed Preparation 2011: An Update." *Advances in Skin & Wound Care* 24(9):415-36, 2011.

PATIENT SCENARIO

Clinical Data

Mrs. AA is 55-year-old female patient who presented with a painful left large toe. She has type 2, adult-onset, non–insulin-dependent diabetes mellitus with neuropathy. There is a palpable dorsalis pedis pulse, and her pain is aggravated by wearing shoes.

The best approach to this patient includes all the components in the WBP paradigm of treating the cause and patient-centered concerns before addressing the components of local wound care (**DIME: D**ebridement, **I**nfection/persistent inflammation, **M**oisture balance, and then the **E**dge effect for advanced therapies if wounds are not healing at the expected rate when all the components of WBP have been corrected).

For patient-centered concerns, the pain that has developed in a neuropathic foot with loss of protective sensation should alert the clinician to the disruption of deeper structures with infection (e.g., osteomyelitis) or a Charcot joint (acute inflamed bony structure due to multiple small pathological fractures).

Case Discussion

For the treatment of the cause, a HbA1c needs to be as ideal as possible for optimal healing. (HbA1c as close as possible to 7—the lower the better and definitely below 12.)

In this case, physical examination revealed a keratotic cap on the left second toe that required debridement. Vascular supply was assessed prior to surgical debridement and was deemed adequate for healing. As is often seen in patients with neuropathy, removal of the surface of the callus revealed encasement of the toenail that had rotated under the tip of the toe with the claw deformity. When the nail and callus were removed, there was a small ulcer that probed to bone. This indicates a high probability of osteomyelitis, which was confirmed with an x-ray showing erosion of the phalangeal bone. Following debridement, a calcium alginate dressing was applied for hemostasis. With this type of dressing, the calcium donated from the dressing assists in local hemostasis while the sodium exchanged for the calcium creates a sodium alginate hydrogel for moist interactive

healing, avoiding a proinflammatory hemorrhagic crust formation (Fig. 8-11).

The treatment principles employed in this case can be remembered with the mnemonic **VIPS**:

- **V**ascular supply with a palpable pulse was adequate for healing (if the pulse is not palpable, an ankle–brachial pressure index, toe pressures, or transcutaneous oxygen determination can be obtained to assess healability).
- **I**nfection: The osteomyelitis was treated with 3 months of oral clindamycin (good bone penetration with gram-positive and anaerobe coverage) combined with ciprofloxacin (gram-negative coverage including *Pseudomonas*), with resolution of the pain and osteomyelitis.
- **P**ressure: The callus was due to pressure in the toe box of the patient's shoes. This was corrected with the ordering of a pneumatic walker (can be made irremovable or treated with a contact cast) with gradual transition to deep-toed shoes and orthotics to remove local pressure.
- **S**harp surgical debridement was performed, including removal of the nail to reveal the osteomyelitis sinus. Maintenance debridement may also be necessary when callus periodically re-forms.

This case illustrates the need for holistic patient care with treatment of cause (VIPS), pain control (acetaminophen with codeine for nociceptive pain and a second-generation tricyclic nortriptyline at night and gabapentin in the daytime), and then local wound care with debridement and the use of calcium alginate followed by a silver alginate dressing.

Show What You Know

1. **Which statement about the purpose of debridement is correct?**

   Debridement:

   A. is not essential for wound healing.
   B. removes debris so cell movement is enhanced.
   C. removes necrotic tissue in order to enhance the wound's biological burden.
   D. reduces the need for moist wound healing.

2. **Which sign in a stable necrotic heel would signal a need for debridement?**

   A. Exudate
   B. Presence of thick, leathery eschar
   C. An impending inspection by a regulator
   D. Yellow slough

3. **Which method is an example of mechanical debridement?**

   A. Collagenases
   B. Maggots
   C. Film dressings
   D. Pulsed lavage

4. **A resident in a long-term care facility is on Coumadin and needs debridement for a necrotic ulcer on his sacrum. Which of the following methods of debridement would be *least* indicated?**

   A. Surgical
   B. Enzymatic
   C. Mechanical
   D. Autolytic

5. **Which method of debridement would be best to use initially for a hospitalized client with an infected large sacral**

pressure injury?

A. Surgical
B. Enzymatic
C. Mechanical
D. Autolytic

Answers to these questions are found on page 683.

References

1. Sibbald, R.G., Elliott, J.A., Ayello, E.A., et al. Optimizing the Moisture Management Tightrope with Wound Bed Preparation 2015©. Advances in Skin & Wound Care 28(10):466-76, 2015.
2. Kirshen, C., et al. “Debridement: A Vital Component of Wound Bed Preparation.” Advances in Skin & Wound Care 19(9):506-17, quiz 518-9, November-December, 2006.
3. Schultz, G.S., et al. “Wound Bed Preparation: A Systematic Approach to Wound Management.” Wound Repair and Regeneration 11(Suppl 1):S1-28, March 2003.
4. Sibbald, R.G., Goodman, L., Woo, K.Y., et al. “Special Considerations in Wound Bed Preparation 2011: An Update©.” Advances in Skin & Wound Care 24(9):415-36, 2011.
5. Ramasastry, S.S. “Chronic Problem Wounds.” Clinical Plastic Surgery 25(3):367-96, July 1998.
6. Wysocki, B. “Wound Fluids and the Pathogenesis of Chronic Wounds.” Journal of Wound Ostomy and Continence Nursing 23(6):283-90, November 1996.
7. Edlich, R.F., et al. “The Biology of Infections: Sutures, Tapes, and Bacteria.” In T.K. Hunt, ed. Wound Healing and Wound Infection: Theory and Surgical Practice. New York: Appleton-Century-Crofts, 1980.
8. Wound, Ostomy and Continence Nurses Society- Wound Guidelines Task Force. WOCN 2016 Guideline for Prevention and Management of Pressure Injuries (Ulcers): An Executive Summary. Journal of Wound, Ostomy and Continence Nursing 44(3):241-6, May/June 2017.
9. Brem, H., et al. “Protocol for Treatment of Diabetic Foot Ulcers.” American Journal of Surgery 187(Suppl 1):1S-10S, 2004.
10. Black, J., Baharestani, M., Black, S., et al. “An Overview of Tissue Types in Pressure Ulcers: A Consensus Panel Recommendation.” Ostomy/Wound Management 56(4):28-44, 2010.
11. European Pressure Ulcer Advisory Panel, National Pressure Ulcer Advisory Panel, Pan Pacific Pressure Injury Alliance. In: E. Haesler, ed. Prevention and Treatment of Pressure Injuries/Ulcers: Clinical Practice Guideline, The International Guideline. Cambridge Media, 2019.
12. Sibbald, R.G., Goodman, L, Woo, KY, et al. “Special Considerations in Wound Bed Preparation 2011: An update. Wound Care Canada 10(2):20-5, 2011.
13. Norton, L, Parslow, N., Johnston D., et al. *Best Practice Recommendations for the Prevention and Treatment of Pressure Ulcers: Update 2017*. Toronto, Canada: Canadian Association of Wound Care, 2017.

14. Niezgoda JA, Cabigas B, Allen H. Managing Pyoderma Gangrenosum: A Synergistic Approach Combining Surgical Debridement, Vacuum Assisted Closure and Hyperbaric Oxygen Therapy. Plast Reconstr Surg 117(2):24e-8e, 2006.
15. Rodd-Nielsen, E., Brown, J., Brooke, J., et al. “Canadian Association for Enterostomal Therapy Evidence–Based Recommendations for Conservative Sharp Wound Debridement.” Journal of Wound Ostomy and Continence Nursing 40(3);246-53, 2013.
16. O’Neill, D.K., Robins, B., Ayello, E.A., et al. “Regional Anaesthesia with Sedation Protocol to Safely Debride Sacral Pressure Ulcers.” International Wound Journal 9:525-43, 2012.
17. Buchnan, K., Bernstein, N., Ayello, E.A., et al. “Cardiac Device Interrogation for Safer Care of Surgical Wound Patients.” Advances in Skin & Wound Care 23(11):507-14, 2011.
18. Yoo, C., Ayello, E.A., Robins, B., et al. “Perioperative Use of Bispectral (BIS) Monitor for a Pressure Ulcer Patient with Locked-in Syndrome (LIS).” International Wound Journal 11(5):540-5, 2014.
19. O’Neil, D.K., Tsui, S., Ayello, E.A., et al. “Anesthesia Protocol for Heel Pressure Ulcer Debridement.” Advances in Skin & Wound Care 25(5):209-19, 2012.
20. Centers for Medicare and Medicaid Services (CMS). “Tag F-314: Pressure Ulcers. Revised Guidance for Surveyors in Long Term Care.” Issued November 12, 2004. Available at: http//cms.gov/transmittals/Downloads/R4SOM.pdf. Accessed October 10, 2014.
21. Scott, R., Loehne, H. “Five Questions and Answers About Pulsed Lavage.” Advances in Skin & Wound Care 13(3, part I):133-4, May-June 2000.
22. Bergstrom, N., Bennett, M.A., Carlson, C.E., et al. Treatment of Pressure Ulcers. Clinical Practice Guidelines, No. 15. AHCPR Publication No. 95-0652. Rockville, MD: Agency for Health Care Policy and Research, December 1994.
23. Mosti, G., Mattaliano, V. “The Debridement of Chronic Leg Ulcers by Means of a New, Fluidjet-based Device.” Wounds 18(8):227-37, August 2006.
24. Leaper, D. “Sharp Techniques for Wound Debridement.” World Wide Wounds. Available at: http://www.worldwidewounds.com/2002/december/Leaper/Sharp-Debridement.html. Accessed July 17, 2006.
25. Williams, D., Enoch, S., Miller, D., et al. “Effect of Sharp Debridement Using Curette on Recalcitrant Nonhealing Venous Leg Ulcers: A Concurrently Controlled, Prospective Cohort Study.” Wound Repair and Regeneration 13:131-7, March-April 2005.
26. Ashworth, J., Chivers, M. “Conservative Sharp Debridement: The Professional and Legal Issues.” Professional Nurse 17(10):585-8, June 2002.

27. Endara M, Attinger, C. “Using Color to Guide Debridement.” Advances in Skin & Wound Care 25(12):549-55, 2012.
28. Brem, H., et al. “Molecular Markers in Patients with Chronic Wounds to Guide Surgical Debridement.” Molecular Medicine 13(1-2):30-9, 2007.
29. Tomic-Canic, M., Ayello, E.A., Stojadinovic, O., et al. “Using Gene Transcription Patterns (Bar Coding Scans) to Guide Wound Debridement and Healing.” Advances in Skin & Wound Care 21(10):487-92, 2008.
30. WOCN. WOCN Conservative Sharp Wound Debridement: Best Practice for Clinicians. Mt Laurel, NJ: WOCN, 2005.
31. Steed, D.L., et al. “Effect of Extensive Debridement and Treatment on the Healing of Diabetic Foot Ulcers. Diabetic Ulcer Study Group.” Journal of the American College of Surgeons 183(1):61-4, July 1996.
32. Kloth L, Niezgoda JA. Ultrasound for Wound Bed Preparation and Healing. In McCulloch JM, Kloth L, eds. Wound Healing: Alternatives in Management, 4th ed. Philadelphia, PA: F. A. Davis, 2011.
33. Lau, Y.S., Brooks, P. “Innovative Use of Povidone-Iodine to Guide Burn Wound Debridement and Predict the Success of Biobrane as a Definitive Treatment for Burns.” Advances in Skin & Wound Care 27(3):111-3, 2014.
34. Wolcott, R.D., Rumbaugh, K.P., James, G., et al. “Biofilm, Maturity Studies Indicate Sharp Debridement Opens a Time Dependent Therapeutic Window.” Journal of Wound Care 19(8):320-8, 2010.
35. Kim PA, Attinger CE. Clinic-based Debridement of Chronic Ulcers Has Minimal Impact on Bacteria. Wounds 30(5):114-9, 2018.
36. Wright, J.B., Shi, L. “Accuzyme Papain-Urea Debriding Ointment: A Historical Review.” Wounds 15(4):2S-12S, April 2003.
37. Falanga, V., Brem, H. Ennis, W.J., et al. “Maintenance Debridement in the Treatment of Difficult-to-Heal Chronic Wounds. Recommendations of an Expert Panel.” Ostomy/Wound Management (Suppl):2-15, June 2008.
38. Ramundo, J., Gray, M. “Collagenase for Enzymatic Debridement. A Systematic Review.” Journal of Wound, Ostomy & Continence Nursing 36(6S):S4-11, 2009.
39. Shi, L., Carson, D. “Collagenase Santyl Ointment. A Selective Agent for Wound Debridement.” Journal of Wound Ostomy & Continence Nursing 36(6S):S12-6, 2009.
40. Smith, R.G. “Enzymatic Debriding Agents: An Evaluation of the Medical Literature.” Ostomy/Wound Management 54(8):16-34, 2008.
41. Hebda, P.A., Lo, C. “Biochemistry of Wound Healing: The Effects of Active Ingredients of Standard Debriding Agents–Papain and Collagenase–on Digestion of Native and Denatured Collagenous Substrates, Fibrin and Elastin.” Wounds 13(5):190-4, 2001.
42. Hebda, P.A., et al. “Evaluation of Efficacy of Enzymatic Debriding Agents for Removal of Necrotic Tissue and Promotion of Healing in Porcine Skin Wounds.” Wounds 10(3):83-96, 1998.

43. Falanga, V. “Wound Bed Preparation and the Role of Enzymes: A Case for Multiple Actions of Therapeutic Agents.” Wounds 14(2):47-57, February 2002.
44. McCallon, S.K., Hurlow, J. “Clinical Applications for the Use of Enzymatic Debriding Ointment and Broad-spectrum Bacteriostatic Foam Dressing.” Journal of Wound, Ostomy & Continence Nursing 36(6S):S17-24, 2009.
45. Hansbrough, J.F., et al. “Wound Healing in Partial-Thickness Burn Wounds Treated with Collagenase Ointment Versus Silver Sulfadiazine Cream.” Journal of Burn Care and Rehabilitation 16(Pt 1):241-7, 1995.
46. Pilcher, B.K., Dumin, J.A., Sudbeck, B.D., et al. “The Activity of Collagenase-1 Is Required for Keratinocyte Migration on a Type I Collagen Matrix.” Journal of Cell Biology 137:1445-57, 1997.
47. Burgos, A., Gimenez, J., Moreno, E., et al. “Collagenase Ointment Application at 24- Versus 48-Hour Intervals in the Treatment of Pressure Ulcers. A Randomized Multicentre Study.” Clinical Drug Investigation 19:399-407, 2000.
48. Muller, E., van Leen, M.W., Bergmann, R. “Economic Evaluation of Collagenase-containing Ointment and Hydrocolloid Dressings in the Treatment of Pressure Ulcers.” Pharmacoeconomics 19:1209-16, 2001.
49. Riley, K.N., Herman, I.M. “Collagenase Promotes the Cellular Responses to Injury and Wound Healing In Vivo.” Journal of Burns and Wounds 4:112-24, July 2005.
50. Mekkes, J.R., et al. “Quantitative and Objective Evaluation of Wound Debriding Properties of Collagenase and Fibrinolysin/Desoxyribonuclease in a Necrotic Ulcer Animal Model.” Archives of Dermatological Research 290:152-7, 1998.
51. Frye, K.E., Luterman, A. “Decreased Incidence of Hypertrophic Burn Scar Formation with the Use of Collagenase, an Enzymatic Debriding Agent.” Wounds 17(12):32-6, December 2005.
52. Alvarez, O.M., et al. “A Prospective, Randomized, Comparative Study of Collagenase and Papain-urea for Pressure Ulcer Debridement.” Wounds 14:293-301, 2002.
53. Mekkes, J.R., et al. “Efficient Debridement of Necrotic Wounds Using Proteolytic Enzymes Derived from Antarctic Krill: A Double-blind, Placebo-controlled Study in a Standardized Animal Wound Model.” Wound Repair & Regeneration 6(1):50-7, January 1998.
54. Bradley, M., et al. “The Debridement of Chronic Wounds: A Systematic Review.” Health Technology Assessment 3(17 Pt 1):iii-iv, 1-78, 1999.
55. Mulder, G.D. “Cost-effective Managed Care: Gel Versus Wet-to-Dry for Debridement.” Ostomy/Wound Management 41(2):68-70, 72, 74 passim, March 1995.
56. Thomas, S., et al. “Clinical Experience with a New Hydrogel Dressing.” Journal of Wound Care 5:132-3, 1996.

57. Trudgian, J. “Investigating the Use of Aquaform Hydrogel in Wound Management.” British Journal of Nursing 9:943-8, 2000.
58. Schimmelpfenning, D., Mollenhauer, S. “Use of a Clear Absorbent Acrylic Dressing for Debridement.” Journal of Wound Ostomy & Continence Nursing 33(6):639-42, November/December, 2006.
59. Konig, M., Vanscheidt, W., Augustin, M., et al. “Enzymatic Versus Autolytic Debridement of Chronic Leg Ulcers: A Prospective Randomised Trial.” Journal of Wound Care 14:320-3, July 2005.
60. Smith, J. “Debridement of Diabetic Foot Ulcers.” Cochrane Database of Systematic Reviews (4):CD003556, 2002.
61. Cuschieri, L., Debosz, J., Miller, P., et al. “Autolytic Debridement of a Large, Necrotic, Fully Occluded Foot Ulcer Using a Hydrocolloid Dressing in a Diabetic Patient.” Advances in Skin & Wound Care 26(7):300-4, 2013.
62. Claxton, M.J., et al. “5 Questions—and Answers—About Maggot Debridement Therapy.” Advances in Skin & Wound Care 16:99-102, 2003.
63. Bolton, L.L. “Evidence Corner, Maggot Therapy.” Wounds 18(9):A19-22, September 2006.
64. Courtenay, M., et al. “Larva Therapy in Wound Management.” Journal of the Royal Society of Medicine 93(2):72-4, 2000.
65. Steenvorde, P., et al. “Maggot Debridement Therapy: Free-range or Contained? An In Vivo Study.” Advances in Skin & Wound Care 18:430-5, October 2005.
66. Chambers, L., et al. “Degradation of Extracellular Matrix Components by Defined Proteinases from the Greenbottle Larva *Lucilia sericata* Used for the Clinical Debridement of Non-healing Wounds.” British Journal of Dermatology 148:14-23, 2003.
67. Wollina, U., et al. “Biosurgery in Wound Healing: The Renaissance of Maggot Therapy.” Journal of the European Academy of Dermatology and Venereology 14:285-9, 2000.
68. Rayner, K. “Larval Therapy in Wound Debridement.” Professional Nurse 14:329-33, February 1999.
69. Margolin, L., Gialanella, P. “Assessment of the Antimicrobial Properties of Maggots.” International Wound Journal 7:202-4, 2010.
70. Sherman, R.A. “Maggot Versus Conservative Debridement Therapy for the Treatment of Pressure Ulcers.” Wound Repair & Regeneration 10:208-14, 2002.
71. Sherman, R.A. “Maggot Debridement Therapy for Treating Non-healing Wounds.” Wound Repair & Regeneration 8:327, 2000.
72. Sherman, R.A., et al. “Maggot Debridement Therapy in Outpatients.” Archives of Physical Medicine and Rehabilitation 82:1226-9, 2001.
73. Tanyuksel, M., et al. “Maggot Debridement Therapy in the Treatment of Chronic Wounds in a Military Hospital Setup in Turkey.” Dermatology 10:115-8, 2005.

Mumcuoglu, K.Y. “Clinical Applications for Maggots in Wound Care.” 74. American Journal of Clinical Dermatology 2:219-27, 2001.
75. Richardson, M. “The Benefits of Larval Therapy in Wound Care.” Nursing Standard 19(7):70, 72, 74 passim, 2004.
76. Thomas, S., Andrews, A., Jones, M. “The Use of Larval Therapy in Wound Management.” Journal of Wound Care 7:521-4, 1998.
77. Sherman, R.A. “Maggot Therapy for Foot and Leg Wounds.” International Journal of Lower Extremity Wounds 1:135-42, June 2002.
78. Wayman, J., et al. “The Cost Effectiveness of Larval Therapy in Venous Ulcers.” Journal of Tissue Viability 10(3):91-4, 2000.
79. Kitching, M. “Patients’ Perceptions and Experiences of Larval Therapy.” Journal of Wound Care 13(1):25-9, 2004.
80. Steenvorde, P., et al. “Determining Pain Levels in Patients Treated with Maggot Debridement Therapy.” Journal of Wound Care 14:485-8, November 2005.
81. Mosher, B.A., et al. “Outcomes of Four Methods of Debridement Using a Decision Analysis Methodology.” Advances in Wound Care 12(2):81-8, March 1999.
82. Saap, L.J., Falanga, V. “Debridement Performance Index and Its Correlation with Complete Closure of Diabetic Foot Ulcers.” Wound Repair & Regeneration 10:354-9, 2002.
83. Falanga, V. “Wound Bed Preparation and the Role of Enzymes: A Case for Multiple Actions of Therapeutic Agents.” Wounds 14(2):47-57, March 2002.
84. Steed, D.L. “Debridement.” American Journal of Surgery 187(5A):71S-4 Review, May 2004.
85. Vowden, K.R., Vowden, P. “Wound Debridement, Part 1: Non-sharp Techniques.” Journal of Wound Care 8:237-40, 1999.
86. Vowden, K.R., Vowden, P. “Wound Debridement, Part 2: Sharp Techniques.” Journal of Wound Care 8:291-4, June 1999.

9

Wound Treatment Options

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We gratefully acknowledge the contributions of Pamela Scarborough, DPT for her work on previous editions of this chapter.

Objectives

After completing this chapter, you'll be able to:

- explain moist wound therapy
- select dressings based on assessment of wound characteristics
- list indications for use of dressings by categories
- state the advantages and disadvantages for each dressing category
- use principles of care in dressing selection
- discuss the use of advanced therapies.

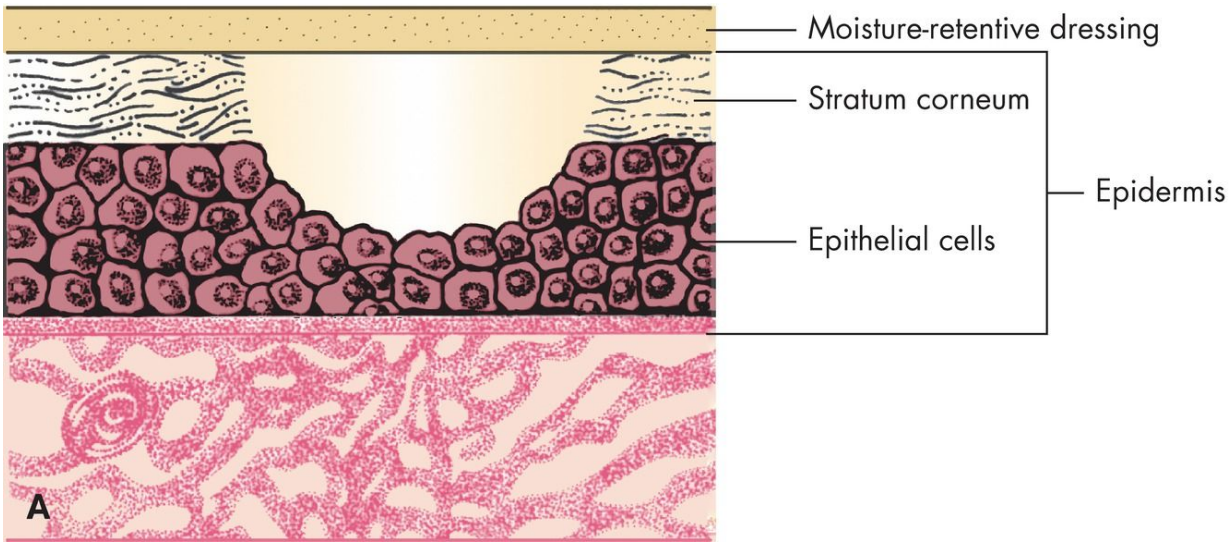
A Challenge for Clinicians

The evidence for the best wound care products to use for wound healing and prevention continues. Although we are gaining new knowledge as to the biology of wound healing, “we can no longer care only for the wound itself; we must step back and look at the entire human being who happens to have a wound that needs healing”.[1] For clinicians, being able to differentiate among the various treatment options, when and how to apply them, in what combinations, and when to change them is both an art and a science. “With the emergence of more complex products, we will be increasingly required to use these products appropriately to maximize their impact. As a better understanding of the wound environment becomes available, our ability to tailor our approach and better treat the patient as a whole increases”.[2] Providing quality care for your wound patients starts with an analysis of the patient’s individualized wound assessment and continues with developing a plan of care, selecting the proper product, and reevaluating the plan of care as appropriate. Wound dressings can present a challenging decision for clinicians. Moist wound healing, moisture-balanced dressings, and certainly the principles of optimal wound interventions are key concepts needed to support the healing process. As clinicians try to heal wounds faster, the marketplace continues to provide many more treatment choices. In 2014, it was reported that more than 500 different types of dressings are available to manage patients with wounds.[3] With the continued evolution of wound dressings, at wound care conferences, that number has risen as reported, to be in the 900s. Keeping abreast of wound dressing choices and various application techniques, as well as which product to use and when, is an ambitious task for all clinicians.

Moist Wound Healing

Wound healing in the 21st century has certainly changed. There have been more advances in wound care over the past four decades than during the previous 2,000 years. This wound care revolution has been due in part to Dr. Winter's discovery (in the 1960s) of the importance of moist wound healing in experimental animals.[4] Hinman and Maibach[5] paralleled these findings of faster resurfacing in partial-thickness wounds in humans. Their research efforts laid the foundation for understanding the importance of moisture and moisture loss in wound healing. The concept of moist wound healing and moisture-balanced dressings is now commonly accepted by most clinicians throughout the world as best practice.

We now understand that wound healing must take place in a moist environment. Epithelial cells require moisture to migrate from the wound edges to re-epithelialize or resurface the wound. This process is likened to "leapfrogging" of the cells. In a dry wound, these cells have to burrow down underneath the wound bed to find a moist area upon which to "march" or move forward (Fig. 9-1).



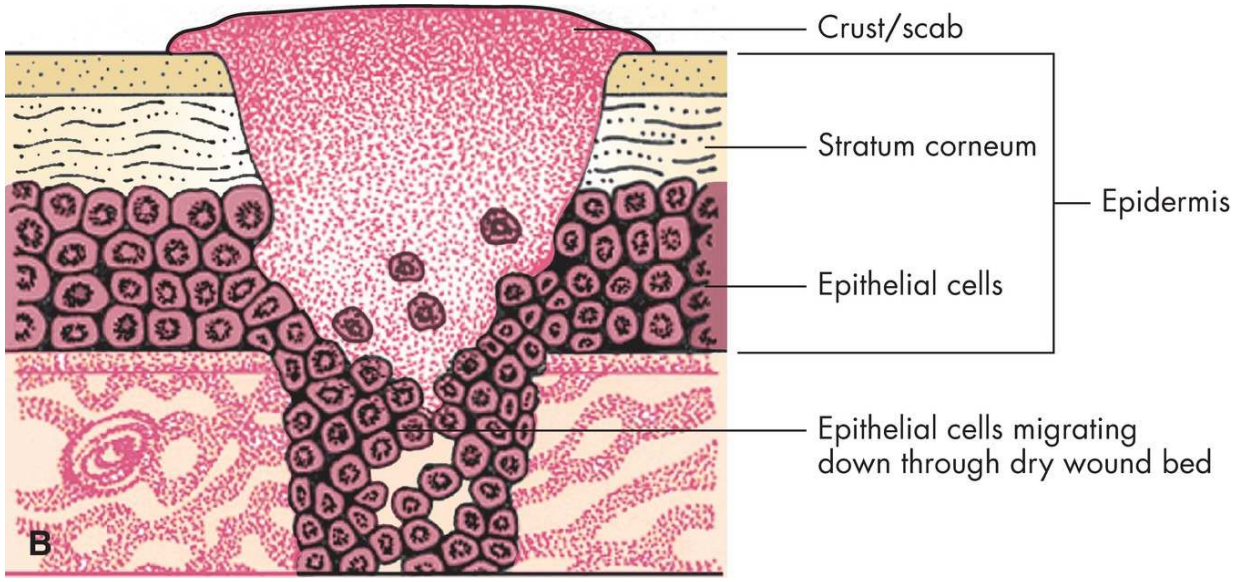


Figure 9-1. Epithelial cell migration in an open wound. **(A)** In a moist environment, epithelial cells can migrate on the wound bed surface to close the wound, as shown. **(B)** In a dry wound bed, epithelial cells burrow underneath the wound bed, as shown.

The concept of moist wound healing based on wound physiology and characteristics required that new dressing materials be developed to replace the passive coverings that had been used in the past, which evolved from such “natural” coverings as feathers, lint, grease, milk, wine, mud, leaves, and other concoctions. Today’s wound dressings are actively involved in stimulating cell proliferation and encouraging epithelial cells to migrate. Moisture-balanced or moisture-retentive dressings also act as a barrier against bacteria and absorb excess wound fluid, creating opportune conditions for healing.[5–8] Formerly, wound dressings were used primarily to protect the wound from secondary infection by forming a barrier against bacteria and absorbing wound fluid. The greatest advantage of contemporary dressings is the maintenance of moist wound conditions, which is in contrast to the “classical gauze techniques” that led to the formation of a dry, firmly adhering scab.[9] Today’s dressings promote rapid healing, act as a barrier, decrease or eliminate pain, require fewer changes,

provide autolytic débridement, and can be cost-effective if used appropriately.

Despite the benefits of newer dressing products, wet-to-dry gauze is still popular with many physicians, especially surgeons who have not been influenced by the development of modern wound dressings.[10] In a study by Pieper and colleagues[11] of 1,638 wounds treated in the home setting, the most commonly used dressing for all types of wounds (*n* = 406) was dry gauze. No dressing (i.e., an uncovered wound) was the second most common treatment (*n* = 252), and saline-moistened gauze (*n* = 145) was third. Advanced moisture-retentive dressings constituted less than 25% of all dressings used. So, despite over half a century of information on the benefits of moist wound therapy, some physicians may still revert back to the "old methods."

Research by Kim et al.[12] suggests that a saline gauze dressing acts as an osmotic dressing. As water evaporates from the saline dressing, it becomes hypertonic. Because the body wants to maintain homeostasis by reestablishing isotonicity, wound fluid is drawn into the gauze dressing. In addition to water, wound fluid includes blood and proteins. These substances form an impermeable layer on the dressing that prevents wound fluid from "wetting" the dressing; the net result is that the dressing dries out. The removal of a wet-to-moist dressing that has dried may then cause reinjury of the wound, resulting in pain and delayed wound healing.[10]

Pain at dressing removal is a frequent complaint heard by patients whose gauze dressings have dried into their wound. In "Principles of Best Practice: Minimising Pain at Wound Dressing-Related Procedures: A Consensus Document," the World Union of Wound Healing Societies recommends that clinicians use dressings that minimize pain and trauma during application and removal.[13]

Gauze dressings don't present a barrier to bacteria. Lawrence[14] demonstrated in an in vitro study that bacteria can pass through up to 64 layers of dry gauze. Once the gauze is moistened, it's even less effective as a barrier against bacteria. Infection rates are higher in wounds in which gauze is used as compared with wounds that are covered with transparent films or hydrocolloids.[15,16]

Successful wound healing depends on maintaining a moist environment. A balanced moist wound environment facilitates cellular growth and collagen proliferation within a healthy noncellular matrix.[17] The right balance of moisture is critical to wound healing. Too much moisture in the wound bed can impair the healing process, damage surrounding skin, and cause periwound maceration. Excess moisture must be managed with an appropriate wound dressing, thereby preventing further tissue destruction and deterioration of the wound bed.[17]

Film and hydrocolloid were among the first dressing materials that could maintain a moist wound healing environment. New application techniques had to be learned and, more importantly, the clinical significance of wound fluid findings understood. The accumulation of light greenish-yellow fluid seen collecting under film dressings has caused us to relearn what is a normal expectation in a healing wound. Even the different nuance of wound odors has been the cause for new learning. For example, different odors occur as wound fluid interacts with different dressing materials. A wound being treated with alginate dressings, which are made from seaweed, may smell like “low tide.”

The new generation of moisture-balanced dressings can maintain the right environment conducive to wound healing. These dressings may have occlusive, semiocclusive, absorptive, hydrating, autolytic, débriding, or hemostatic characteristics. Various wound dressing types will be discussed later in the chapter.

Treatment Decisions

The myriad of products available for wound care have enhanced the overall management of patients, but they have also created confusion about selecting the appropriate product. Optimal wound interventions should be dependent on the basic principles of wound care, attentive wound assessment, and expected outcomes. A complete wound assessment should be the driving element in all treatment decisions. (See Chapter 6, Wound Assessment.) Wound assessment should be based on the principles of wound care (Table 9-1).

Table 9-1 Principles of Care: The MEASURES Acronym

Minimize trauma to wound bed
Eliminate dead space (tunnels, tracts, undermining)
Assess and manage the amount of exudates
Support the body's tissue defense system
Use nontoxic wound cleansers
Remove infection, debris, and necrotic tissue
Environment maintenance, including thermal insulation and a moist wound bed
Surrounding tissue, protect from injury and bacterial invasion

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Wound treatment decisions must be patient centered. What are the patient's goals and preferences? Local wound care starts with a thorough assessment of the wound and a comprehensive collection of data about the patient's overall status. Wound assessment parameters can assist with treatment choices and decisions for appropriate dressing selection[18] (Fig. 9-2).

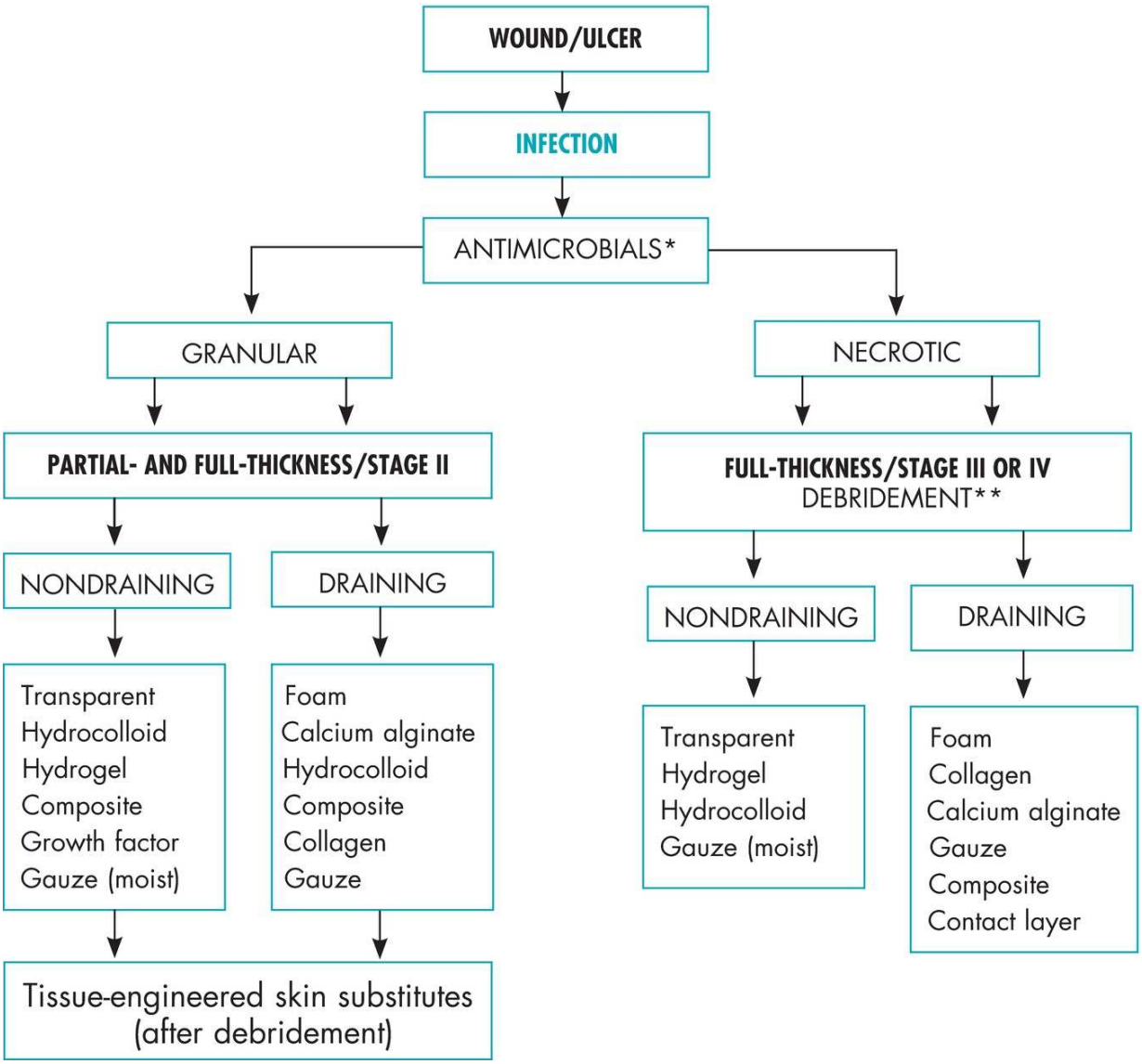

Figure 9-2. Wound care decision algorithm. Algorithm isn't inclusive of all available products. Always read package insert before use; product use may vary.

*Antimicrobials may be topical or within a dressing component. **Débridement may be done by surgical, mechanical, autolytic, or enzymatic methods—products vary. (© Baranoski, S., McIntosh, A., Montoya, L. Optimizing the use of Dressing, Lecture, Clinical Symposium on Advances in Skin & Wound Care. Las Vegas, NV, September 2014.)

Once a thorough, individualized wound assessment is complete, choosing dressings and treatments becomes a clinical decision that is mutually arrived at with the patient based on data collected during the assessment and the overall expected outcome.

Practice Point

Dressing choices = Wound assessment + Principles of wound care

Treatment goals may aim to achieve a clean wound, heal the wound, maintain a clean wound bed, or place the patient in another setting to continue care. Clinicians should match the wound assessment characteristics with the dressing characteristics or function. The goal of care then becomes *using the right product on the right wound at the right time*. For example, a granular, nondraining moist or wet wound needs to maintain a moisture balance that is conducive to healing. The primary dressing choice would be a product that maintains a moist environment but doesn't cause maceration or desiccation of the wound bed. In another example, the goal of dressing selection for a necrotic draining wound is to loosen or soften the eschar for surgical débridement or to assist in autolytic débridement of the wound (see

Chapter 8, Wound Débridement), absorb the excess exudate, and prevent trauma to surrounding tissue.

Practice Point

If the wound is dry, add moisture. If the wound has drainage, absorb it. If the wound has necrotic tissue, débride it.

Clinicians need to reassess the wound status when completing dressing changes so that appropriate treatment interventions can be implemented. It's important to also understand that once the characteristics of the wound assessment change, so may the dressing choice. All wound products come with product information and instructions to guide the user in appropriate use of that product. The most appropriate dressing should be selected based on consideration of the patient, the wound, and the site[19] (Tables 9-2 and 9-3).

Table 9-2 Characteristics of an Ideal Dressing

Use the following characteristics to determine the ideal dressing for your patient.

The ideal dressing should:

- maintain a moist environment
- facilitate autolytic débridement
- be conformable for the range of use needed (such as to fill tunneling, undermining, or sinus tracts to eliminate dead space)
- come in numerous shapes and sizes
- be absorbent
- provide thermal insulation
- act as a bacterial barrier
- reduce or eliminate pain at the wound site; pain-free removal.

The following considerations can be used to evaluate the dressing:

- Number of days the dressing can remain in place
- Reason for change or removal
- Appearance of dressing (soiled or intact)
- Ease of dressing application
- Ease of dressing removal
- Ease of dressing maintenance
- Ease of teaching about dressing to caregiver

Adapted from Seaman, S. "Dressing Selection in Chronic Wound Management." *Journal of the American Podiatric Medical Association* 92(1):24-33, January 2002. Reprinted with permission from JAPMA.

Table 9-3 NICE© for Dressing Decision Making

There are thousands of dressings available, and the clinician needs to decide which dressing to select for a particular wound. Ask yourself the following questions about the wound to help determine the dressing that is NICE© to use:

- Is there any **N**ecrotic tissue that needs to be débrided? (Make sure the wound has the ability to heal; if not, however, moist interactive dressings and active surgical débridement to bleeding tissue are contraindicated.)
- Is the wound **I**nfected or inflamed? (Clinicians often look for more than one sign or symptom before diagnosing infection.)
- Do the specific wound **C**haracteristics, such as location, need to be considered? (If the wound is around the anus, a waterproof adhesive dressing may be preferred.) Is pain an issue?
- Is there any **E**xudate; if so, why, how much, and what are the color and consistency?

Exudate may indicate that the cause of the wound has not been treated (e.g., edema due to venous insufficiency), that congestive heart failure is present (look for bilateral involvement and extension above the knee), that albumin levels are low (malnutrition, kidney, or liver disease), or that infection is present. Periwound skin needs protection from exudate by using absorbent dressings and protecting the periwound skin. Select a dressing by answering the four questions. Remember, it's NICE© to pick the right dressing.

Letter	Key Information to Know	Caution
Necrotic tissue slough, eschar	• Wet-to-dry dressings are a nonselective method of mechanical débridement. • Autolytic débridement of tissue is best accomplished with hydrogels, hydrocolloids, and alginate dressings. • With dressing-stimulated autolytic débridement, watch for secondary infection and remove unwanted slough with dressing change.	• Dressings are a slower method of débridement compared with sharp/surgical methods. • There is limited use of wet-to-dry dressings as a débridement method. • Some dressings cannot be used or caution is urged in necrotic wounds; check with the manufacturer for any contraindications for use. • Removal of nonviable tissue is a critical step in preparing the wound bed for healing.
Infection/ inflammation	• Consider using antimicrobial dressings (e.g., silver or iodine). • Infected wounds may require more frequent dressing changes.	• Not all dressings can be used in infected wounds; check with the manufacturer for the specific brand indicated for use.
Characteristics	• Select and reassess a dressing based on location of the wound, such as the use of conformable dressings for hard-to-fit areas. • Waterproof dressings may be used if incontinence is an issue. • Consider the patient's pain, and select dressings that may promote comfort and pain reduction.	• Change dressings when they become soiled from feces or urine. • Different dressings can remain in place for different lengths of time; check with the manufacturer for recommended frequency for dressing changes. • Avoid dressings that may increase or contribute to wound pain and consider systemic pain management strategies.
Exudate	• Match the absorbency of the dressing (none, low, moderate, heavy) to the amount of exudate from the wound. • Assess surrounding skin to evaluate for macerations.	• Surrounding skin needs to be protected from wound drainage; refer to the enabler LOWE. Search for the cause of the excessive exudate and the need to correct the cause. Exudate may be an indicator of infection.

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Practice Point

Wound dressings should be changed to meet the characteristics of the wound bed.

Practice Point

Read and understand the information in the package insert before using a wound care product. Not all wound dressings will function in the same way.

Dressings that come in contact with the wound bed are considered primary dressings. Primary dressings fall within three categories: those that maintain adequate moisture, those that absorb excess moisture, and those that add moisture.[9] Secondary dressings are those that cover a primary dressing or secure a dressing in place. Clinicians should know which dressings are safe to be put into the wound itself and which are used as securement products. Several dressings on the market act as both primary and secondary dressings. Again, what wound characteristics are you addressing?

Dressing selections should also include an assessment of the patient's outcome of care. High-priced, inappropriate dressings are often used when a more cost-effective product would suffice. Outcome is commonly driven by institutional setting. Acute care patients whose length of stay is 4 to 5 days usually won't achieve healing as their outcome, but they will achieve a moist, clean wound bed that supports the healing environment. Home care and long-term care settings may have a goal of healing or maintaining the current

status of the wound based on the overall health status of the patient. Wound outcomes need to be patient-focused and realistic with regard to the length of time a patient is cared for.

The clinician also needs to keep in mind that one of the primary goals of care is the prevention of wound-related infection. Infection is a common complication of all open wounds. Open wounds are colonized with bacteria, which means that low numbers of bacteria are always present on the wound surface.[20] Wounds that are critically colonized can be managed with antimicrobial or antiseptic dressings that provide sustained release of various agents, such as silver or cadexomer iodine.[21] If a wound fails to respond, the clinician should consider switching to a product with a different mechanism of action.[20] Reviewing the NICE model for dressing selection can help streamline your decision process.[17] (See Chapter 7, Wound Bioburden and Infection.) The practice of using the same wound dressing during the entire healing period is no longer valid. *All* wounds under the care of clinicians should be assessed a minimum of once a week and more often if notable changes occur. The type of wound, status of the wound, clinical setting, and regulatory compliance may dictate a different interval of assessment, however. Be aware of practice regulations for your facility or institution. (See Chapter 6, Wound Assessment.)

Wound assessment is the cumulative process of observing the wound itself as well as observing the patient and collecting and evaluating data. For many patients, weekly reassessment will provide the indices of successful treatment and guide decisions that suggest product changes. As the wound characteristics change, so too should the choice of the wound dressing. Indeed, several different types of products may be needed as the wound progresses through the stages of healing.

Dressings should be matched carefully to the wound, the patient, and the setting. For example, a deep wound with a large amount of drainage will require a highly absorbent dressing such as alginate or foam. As the depth and amount of drainage decrease, a dressing such as a hydrogel, hydrocolloid, or film might be used.

Over the course of healing, the treatment plan will change as the wound is filled with granulation tissue and epithelialization occurs. Economic factors should also be considered when selecting dressings. The overall cost and effectiveness of treatment, cost of materials, nursing time, and frequency of dressing changes over time all impact the economic burden of care[22,23] (Table 9-4).

Table 9-4 Economic Considerations for Nurses

- Clean, rather than sterile, dressings and gloves can usually be used in the home for chronic wounds (refer to your agency's policy).
- Saline solution can be made at home by adding one teaspoon of salt to one quart of boiling water.[21]
- Dressings shouldn't be left open or at the patient's bedside.
- Cost of product selected and resources available for financial assistance should be considered.
- Frequency of dressing changes and cost-effective use of materials should be considered.
- Fistula management may use pouching versus dressing.
- Nursing time per dressing change should be considered.
- The overall effectiveness of the treatment should be an economic consideration.

Adapted with permission from Baranoski, S., McIntosh, A., Montoya, L. Optimizing the use of dressing. Lecture, Clinical Symposium on Advances in Skin & Wound Care. Las Vegas, NV. September 2014.

The notion that all wounds are alike has also changed. An understanding of the etiology of the wound is essential for appropriate care.[21] Local wound care products as well as supportive care must be individualized for the particular wound. For example, a venous stasis ulcer might require a highly absorptive dressing as well as the necessary compression therapy. A variety of two- to four-layered compression bandages beyond the classic “Unna boot” are now available. Further, checking for ankle–brachial index and/or toe pressures using a Doppler is part of the total care of a patient with a peripheral vascular ulcer or history of diabetic neuropathies (see Chapters 15, 17, 18).

Using Wound Healing Biology to Select Treatment

An example of increased understanding of the “cellular biology” of wound healing and technology is the use of growth factors in wound care. All growth factors are proteins that are secreted by cells and have the ability to stimulate cell division, a positive action during the wound healing process.[24] Growth factors are now available derived either from a patient’s own platelets or in a drug form dispensed in a tube to be applied to certain wounds. Research continues as to what combination, what quantity, and when growth factors will best enhance wound healing.

Yet another way technology is providing new options for wound management is in the use of tissue-engineered skin equivalents or substitutes for healing chronic wounds.

What the future holds for the use of gene therapy in wound healing is yet to be seen. The use of gene delivery or microencapsulation in advanced wound therapy is being investigated.[24]

Moist Wound Therapy and Dressing Options

The essential function of a wound dressing is to provide the right environment to enhance and promote wound healing. Research over the past 50 years has led to the generally accepted phenomenon that moist wound dressings create an optimal environment for wounds to heal faster and with less scar formation. The work of Orland[25] and Winter[4] led to the development of moist wound dressings as a clinical intervention to treat wounds.

George Winter is often cited as the father of moist wound healing. His laboratory work comparing the effect of air drying versus occlusive dressings on epithelialization in the animal model is generally considered a landmark study.[4,26] Fear of increasing infection with occlusive therapy slowed the development of moist wound therapy dressings, however, and 16 years passed between Winter's study and the development of what is considered the first moist wound therapy dressing, OPSITE. Continued research, clinician experiments, and interest in wound healing in general have led many companies to develop an assortment of moist wound treatment dressings. However, this has created a challenge for healthcare providers, who struggle to keep up with the ever-increasing number of new products and new technologies.

The following synopsis reviews the major dressing categories and provides helpful practice points on what, when, and how to use these dressings.

Transparent Film Dressings

Transparent film dressings, so named for their "see-through" properties, are thin polyurethane membranes. They are coated with an adhesive that allows them to adhere to the wound margins without sticking to the actual wound (Fig. 9-3).

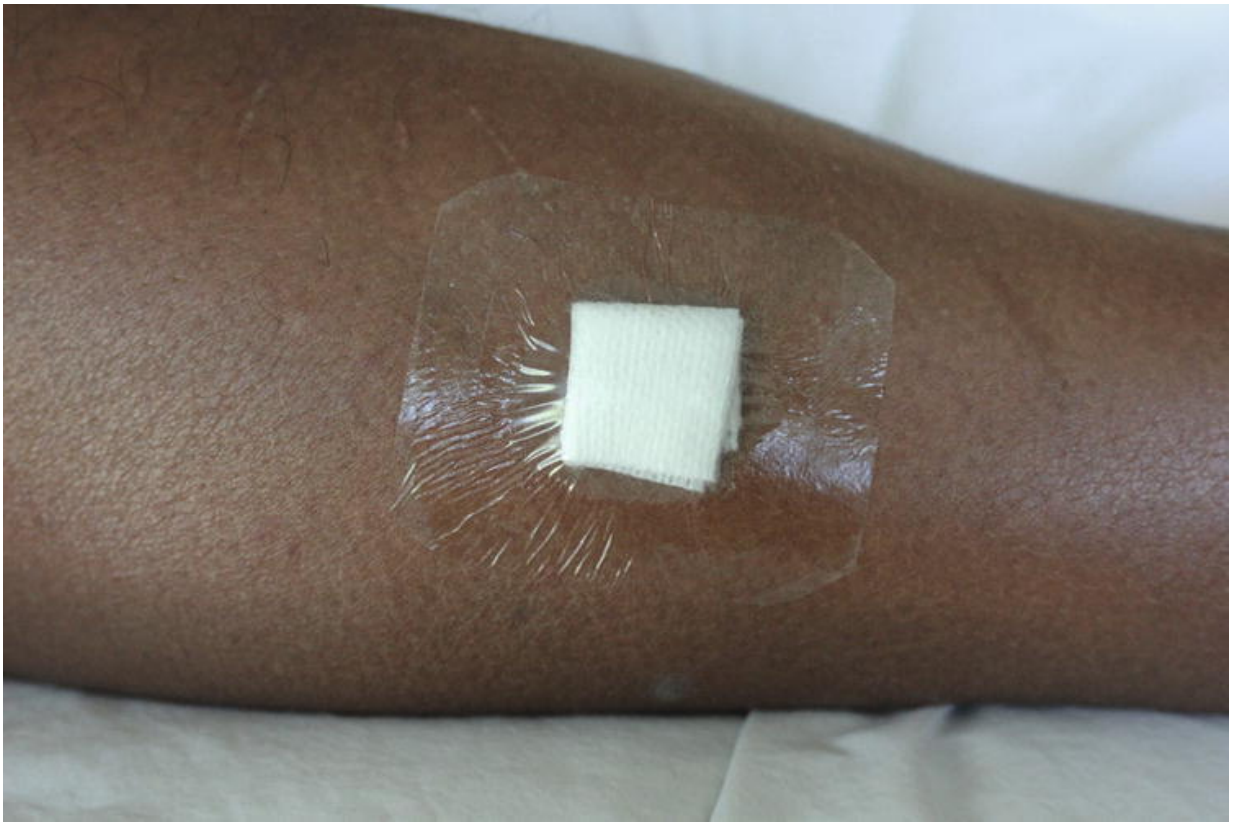

Figure 9-3. Transparent film dressing. (Photo courtesy of L. Montoya.)

Transparent films have no absorptive capacity but do transmit moisture vapor and are semipermeable to gases. These dressings imitate the outer skin layer to provide a moist environment, similar to a blister. This covering allows epithelial cells to migrate over the surface of the wound. Fluid may accumulate under these dressings. This fluid is sometimes mistaken for pus, a sign of infection. The fluid is a useful adjunct to create an autolytic environment, thereby inducing a cleaner wound surface. When excess fluid accumulates or leaks out from the sides of the dressing, the dressing needs to be changed. Maceration of periwound skin can occur if these dressings are not changed in a timely manner.

Transparent film dressings provide a valuable protective barrier against outside contaminants, fluid, and bacteria. Transparent films also add a layer of protection to the wound bed to minimize further

damaging trauma.[17] They provide protection from friction and aid in autolytic débridement. Film makes an excellent secondary dressing as well. Most films can be left on for up to 7 days. These dressings are indicated for wounds with absent or low levels of exudate.[7,8,17] Transparent films can be used on a variety of wound types, such as superficial pressure injuries, minor burns, or lacerations; over sutures, catheter sites, and donor sites; and for protection of the skin against friction. Transparent dressings can be used on central lines and peripherally inserted central catheter lines. Some of the transparent films contain ionic silver for infection prevention.

Practice Essentials

- Apply transparent film dressings to healthy skin; use with caution on aging and fragile skin.
- These dressings may be used on dry to minimally draining wounds; not recommended for skin tears.
- Transparent film dressings make excellent secondary dressings.
- When removing the dressing, lift the corner and pull the film toward the outside of the wound to break the adhesive barrier. Gently stretch the corner of the dressing and support the skin as you remove the dressing.
- Skin protective wipes and sprays can be used on the periwound area to provide an additional seal to prevent the dressing edges from rolling.

Practice Point

Transparent film and hydrocolloid dressings are known for their ability to maintain existing moisture levels in the wound.

Hydrocolloid Dressings

Hydrocolloid dressings were introduced in the 1980s and were the mainstay for wound management for many years. Hydrocolloids are impermeable to gases and water vapor[27] and are composed of opaque mixtures of adhesive, absorbent polymers, pectin gelling agents, and sodium carboxymethylcellulose (Fig. 9-4). Hydrophilic particles within the dressing react with the wound fluid to form a soft gel over the wound bed. According to Choucair and Phillips,[27] some hydrocolloid dressings provide an acidic environment and some act as a bacterial or viral barrier. Their translucent appearance allows for viewing of the amount of exudate absorbed and fluid accumulated under the dressing.

Figure 9-4. Hydrocolloid dressing. (Photo courtesy of A. McIntosh and L. Montoya.)

Hydrocolloid dressings may have a noticeable odor during dressing changes. This is normal in the absence of clinical signs of infection. Some hydrocolloids may also leave residue in the wound bed.

Hydrocolloid dressings are available in many shapes, sizes, and forms (sheet, paste, powder) that fits most wounds and locations. Adhesive properties and ability to absorb exudate vary by product. Most of these dressings are adhesive, so care must be taken when using on fragile skin. Correct application requires the dressing to be bigger than the actual wound size. For optimal dressing adherence, the dressing must extend at least 1 inch (2.5 cm) onto the healthy skin surrounding the wound. The dressing should be changed as recommended by the manufacturer. This could be from 3 to 7 days and depends on the amount of exudate. Some hydrocolloids have other absorptive ingredients added such as alginate and collagen.

Hydrocolloids are indicated for minimally to moderately heavy exudating wounds, abrasions, pressure injuries, dermal wounds, granular wounds, or necrotic wounds. Hydrocolloids do not absorb fluids rapidly, which is valuable when wound exudate levels are in transition from higher to lower quantities.[9] Hydrocolloids also provide a moist environment that is conducive to autolytic débridement. Excessive granulation (hypergranulation tissue) and maceration can occur if the dressing isn't changed appropriately. Hydrocolloids are often used as a preventive dressing on high-risk areas (sacrum, heels) and around surgical wounds to protect the skin from frequent tape removal.

Practice Essentials

- Change the dressing every 3 to 7 days or before it reaches its maximal absorption or when fluid reaches within 1 inch of the edge.
- These dressings are not recommended for undermining, tunnels, or sinus tracts.
- Hydrocolloid dressings may be cut to fit the wound area, such as on an elbow or heel.
- These dressings may be used as primary or secondary dressings. Skin protective wipes or sprays may be used on the periwound area to enhance adherence.

- Picture-framing with paper tape may prevent the dressing edges from rolling.

Hydrogel Dressings

Hydrogel dressings have provided clinicians with a viable means to hydrate or donate moisture to dry wound beds.

Hydrogel dressings are marketed in multiple formats containing varying amounts of water in various polymer matrices. Hydrogels are available as amorphous gels, three-dimensional sheets, or amorphous gels impregnated into other mesh-type dressings (Fig. 9-5). Their unique cross-linked polymer structure entraps water and reduces the temperature of the wound bed by up to 5°C.[27] This moist environment facilitates autolysis and removal of devitalized tissue.

A

Figure 9-5. (A) Hydrogel dressing. **(B)** Amorphous hydrogel dressing. (Photos courtesy of S. Baranoski, A. McIntosh, and L. Montoya.)

The main application for hydrogels is hydrating dry wound beds and softening and loosening slough and necrotic wound debris. Hydrogels have a limited absorptive capacity due to their high water concentration. Some hydrogels have other ingredients, such as alginates, collagen, or starch, to enhance their absorptive capacity and will absorb low to moderate amounts of exudate. They can be used for many types of wounds, including pressure injuries, partial- and full-thickness wounds, and vascular ulcers. Their soothing properties also make them excellent choices for use in skin tears, dermabrasion, dermal wounds, donor sites, and radiation burns.

Maceration can be a concern for clinicians. Periwound skin areas need to be protected from excess hydration; therefore, protective barriers are often recommended. One of the benefits of a hydrogel is that it can be used with topical medications or antibacterial agents. Hydrogels are packaged as sheets, tube gels, sprays, and impregnated gauze pads or strips for packing tunneling and undermined areas within the wound bed. Some require a secondary dressing to secure the hydrogel; new versions have adhesive borders. Some newer versions also contain ionic silver and can be left on for several days. Hydrogels with silver should not be used in combination with enzymatic débriding agents because the silver can inactivate the enzyme.

Practice Essentials

- Do not use hydrogels on wounds with heavy exudate. Daily dressing changes may be necessary due to evaporation of the hydrogel. Some sheet hydrogels may last for several days.
- Protect the surrounding skin with a skin barrier ointment, wipe, or spray.
- Monitor for areas of maceration on surrounding skin.

Honey (Active Leptospermum)

Honey dressings are composed of medical-grade active *Leptospermum* or Manuka honey. These products have multiple mechanisms of action including reducing edema, lowering wound PH, and promoting autolytic débridement. They are indicated for infected or highly colonized wounds and wounds with slough or necrosis. It is also indicated for partial- and full-thickness wounds and venous and diabetic ulcers. These products are available in various forms. Depending on the amount of exudate, different types of dressings may be selected. Products are available in gel, sheets, contact layers, and as a hydrocolloid (Fig. 9-6).

Figure 9-6. Honey dressing. (Photo courtesy of S. Baranoski, A. McIntosh, and L. Montoya.)

Honey dressings are indicated for infected or highly colonized wounds and wounds with slough or necrosis. They are also indicated for pressure injuries, partial- and full-thickness wounds, and venous and diabetic ulcers. Honey dressings are contraindicated in patients allergic to honey and may have a brief stinging sensation.

Practice Essentials

- Honey products lower the pH of the wound, which may cause a stinging sensation in some individuals.
- These dressings can be used to promote autolytic débridement.
- They may require a secondary dressing.

Foam Dressings

Foam dressings are highly absorbent and are generally made from a hydrophilic polyurethane base[28] (Fig. 9-7). Foam dressings are permeable to both gases and water vapor. Their hydrophilic properties allow for absorption of exudate into the layers of the foam.

Figure 9-7. Foam dressing. (Photos courtesy of A. McIntosh and L. Montoya.)

Foam dressings are very versatile and available in multiple size and shape options. They are indicated for wounds with moderate to heavy exudate, prophylactic protection over bony prominences, or friction areas. Foams can be a primary or secondary dressing for partial- and full-thickness wounds. They are an effective choice for use with

granular wounds, skin tears, and donor sites; under compression wraps; and with hypergranulation tissue. They can also be used on infected wounds and can be left on for 3 to 5 days, depending on product and exudate.[19] Their absorption capability depends on the thickness and composition of the dressing. Foams are also available with controlled-release ionic silver. These dressings must have exudates for the silver ion to be released.

Foams are obtainable with or without an adhesive border, and some are available with a transparent film coating that acts as a bacterial barrier. Sacral foams are often used proactively for prevention. Foams may be used in combination with topical treatments and/or enzymatic débrider.

Practice Essentials

- Foam dressings can be left in place for up to 3 to 5 days, depending on the amount of exudate. Dressing removal is usually trauma free.
- Skin wipes or sprays can be used to protect the periwound area from maceration.
- Sacral foam dressings should be considered prophylactically for the prevention of pressure injuries.
- Determine the *correct* side of the foam is in contact with the wound bed.

Calcium Alginate Dressings

Calcium alginate dressings provide yet another choice for clinicians to use in managing highly exudative wounds (Fig. 9-8). Alginate dressings are absorbent, nonadherent, biodegradable, nonwoven fibers derived from brown seaweed, composed of calcium salts of alginic acid and mannuronic and guluronic acids.[27,28]

Figure 9-8. Calcium alginate dressing. (Photo courtesy of S. Baranoski, A. McIntosh, and L. Montoya.)

When alginate dressings come in contact with sodium-rich solutions such as wound drainage, the calcium ions undergo an exchange for the sodium ions, forming a soluble sodium alginate gel. This gel maintains a moist wound bed and supports a therapeutic healing environment. Alginates can absorb 20 times their weight; this may vary based on the particular product. They are extremely beneficial in managing large draining wounds, pressure injuries, vascular ulcers, wound dehiscence, tunnels, sinus tracts, and infected wounds. Additionally, their hemostatic and absorptive properties make them useful on minor bleeding wounds. Alginates are contraindicated for dry wounds, eschar-covered wounds, and third-degree burns.

Alginates come in numerous sizes and are available in sheet, pad, and rope forms. Some calcium alginate dressings also contain controlled-release ionic silver; these calcium alginate–silver dressings should not be used in combination with enzymatic débriding agents. Alginate dressings are usually changed daily or as indicated by the amount of drainage. One disadvantage is that these dressings can dry out and adhere to the wound bed. Using appropriate secondary dressings that help to maintain a gelling state is recommended.[9]

Practice Essentials

- Calcium alginate dressings provide easy application and trauma-free removal.
- This is a good option for moderate to heavy draining wounds.
- Calcium alginate dressings require a secondary dressing.
- This product is absorptive; therefore, it should not be moistened prior to placing in the wound bed.
- Educate the patient and caregiver that the “gel” formed is normal and not a sign of infection.
- This product may leave fiber residue, which can be removed by flushing with saline.
- Calcium alginate dressings facilitate autolytic débridement.
- These dressings can be used on infected wounds, with or without ionic silver.
- This is a dressing choice for undermining and/or tunneling wounds.

Practice Point

In order to release the silver ion, moisture/exudate must be present when controlled-release ionic silver is used in combination with other types of dressings (hydrocolloids, foams, etc.).

Composite Dressings

A combination of materials makes up a single-layered composite dressing. These dressings have multiple functions, such as a bacterial barrier, absorptive layer, foam, hydrocolloid, or hydrogel.[28] Additionally, they must have an adhesive border and semiadherent or nonadherent properties. These dressings are conformable and are available in numerous sizes and shapes.

However, not all composite dressings provide a moist environment; many are used or created by using a secondary dressing. They are also referred to as *island dressings*.

Practice Essentials

- Use composite dressings with caution when treating a patient with fragile skin.
- These dressings may be used on infected wounds and with topical products.
- Frequency of dressing change depends on the wound exudate and the manufacturers' guidelines.
- These dressings may adhere to wound bed; use caution when removing them.

Collagen Dressings

Collagen dressings should be considered when the wound has failed to progress through the normal wound healing process. This is commonly the result of an extended inflammatory phase, often caused by matrix metalloproteases (MMPs) in the wound. MMPs are necessary to break down damaged tissue; however, too many can destroy the healthy extracellular matrix (ECM) and impede wound healing. When delayed wound healing occurs, collagen dressings can restart the wound healing cascade.[29]

Collagen dressings support a moist wound healing environment, encourage the deposition of new collagen fibers, and support new tissue growth and granulation tissue in the wound bed.[30]

Collagen is a major protein of the body and is necessary for wound healing and repair. Collagen dressings are derived from

bovine, ovine, porcine, equine, or avian. Collagen dressings either are 100% collagen or may be combined with alginates or other products. They are a highly absorptive, hydrophilic, moist wound dressing (Fig. 9-9).

Figure 9-9. (A) Collagen dressing. **(B)** Collagen moistened dressing (Photo courtesy of A. McIntosh and L. Montoya.)

Seaman[19] suggests that collagen powders, particles, and pads are useful in treating highly exudative wounds. If the wound has low to moderate exudate, sheets should be used. If the wound is dry, gels should be used. Collagen dressings can be used on granulating or necrotic wounds and on partial- or full-thickness wounds.[19] They may be used with other topical agents.

A collagen dressing should be changed every 1 to 3 days depending on manufactures directions. Collagen dressings require a secondary dressing for securement.

Practice Essentials

- Collagen dressings are most effective when used on granular tissue.
- These dressings are contraindicated for patients who are sensitive to bovine, ovine, porcine, equine, or avian products.
- Do not use these dressings on dry wounds or third-degree burns.
- Their gel properties prevent these dressings from adhering to the wound bed.
- Collagen dressings facilitate a moist wound environment and may be used with other topical products.

Contact Layer Dressings

Contact layer dressings are a single layer of a woven net that acts as a low-adherence material when applied to wound surfaces.[28] A contact layer dressing is applied directly to the wound and acts as a protective interface between the wound and the secondary dressings. Their main purpose is to allow exudate to pass through the contact layer and into the secondary dressing. The secondary dressing is changed more frequently. The contact layer can be left in place up to 7 days to prevent trauma and pain to the tissue of the wound bed. They are often used with ointments, creams, or other topical products. Contact layer dressings are not recommended for dry wounds or third-degree burns. Check the package insert for clarification as to which wounds the product can be used on. Various sizes and shapes are available.

Practice Essentials

- Contact layer dressings are easy to apply and are secured with a secondary dressing.
- Contact layer dressings are not recommended for dry wounds or third-degree burns.
- Frequency of dressing change depends on the amount of exudate.
- May be beneficial when used directly on the wound with negative pressure wound therapy (NPWT) for pain-free removal.

Antimicrobial Dressings

Antimicrobial dressings have added another dimension to the wound dressing arena. These dressings are different than topical antibiotic therapy. They provide the benefit of an antimicrobial effect against bacteria and a moist environment for healing. The active ingredient may be silver ions, cadexomer iodine, honey, or polyhexamethylene biguanide (PHMB) (Fig. 9-10). Antimicrobial dressings do not replace the need for systemic antibiotic therapy; rather, they serve as an adjunct in treating wound infections. Antimicrobial dressings are available in a variety of forms: transparent dressings, alginates, contact layers, gauze, island dressings, foams, and absorptive fillers. Some of these dressings can remain in place for several days.

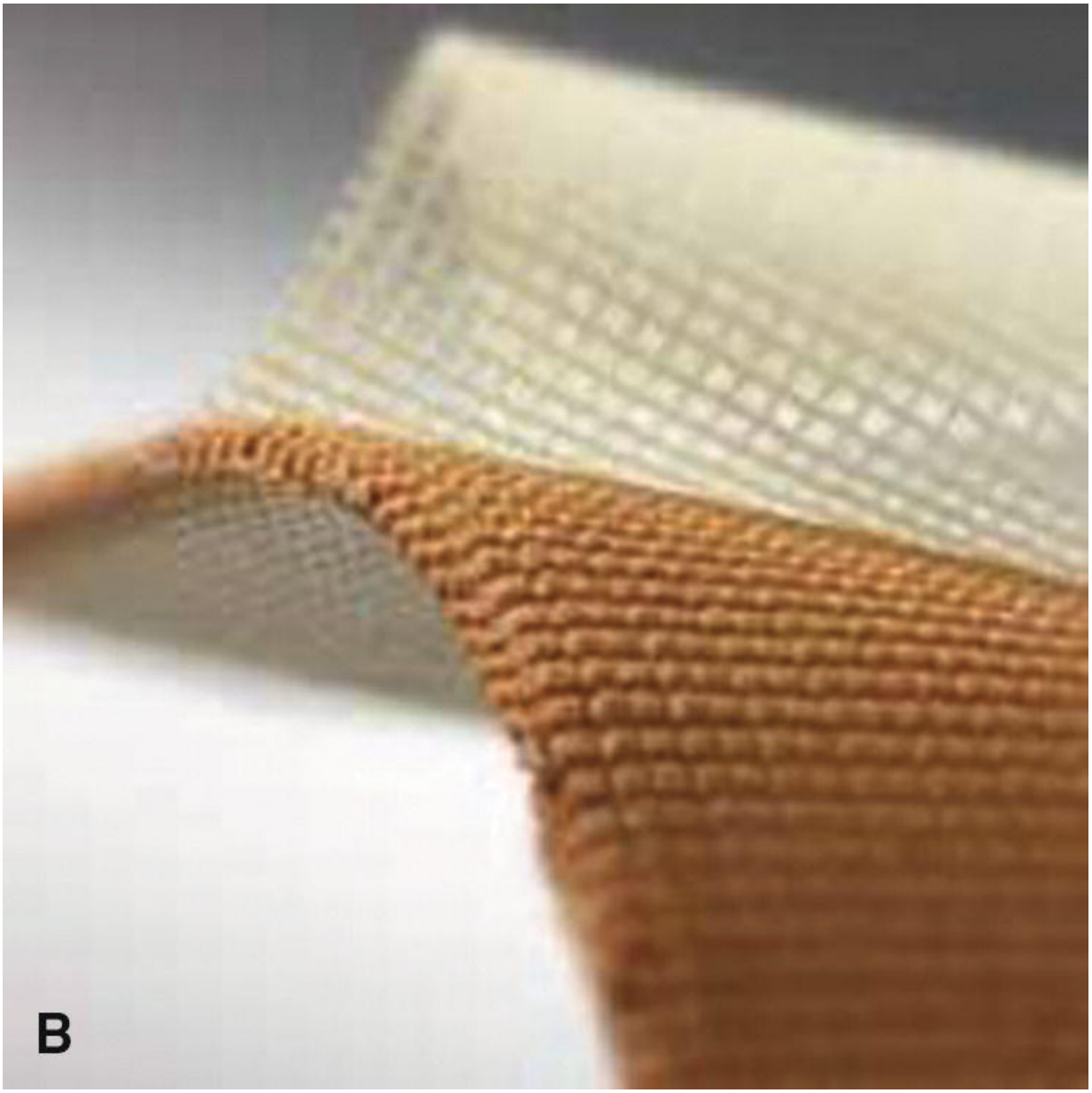

Figure 9-10. Antimicrobial dressings. **(A)** Silver dressing. **(B)** Cadexomer iodine dressing. (Photos courtesy of A. McIntosh and L. Montoya.)

Silver dressings are indicated to reduce bioburden in colonized or infected wounds. Their action depends on the characteristics and composition of the product. According to Sussman,[31] "The level of silver content in dressings varies greatly. The mode of action also

varies; that is, some release silver into the wound, others partly release the silver and hold some in the dressing, and others keep the silver within the dressing."

Practice Essentials

- Antimicrobial dressings are an adjunct in treating wound infections.
- Some silver antimicrobial dressings may cause temporary stain of the skin.
- Educate patient that the silver dressing must be removed prior to an x-ray, MRI, or radiation.
- Silver antimicrobial dressings should not be used with an enzymatic derider, as it will inactivate them.

Absorptive Dressings

Absorptive dressings can be classified based on their degree of absorbency and occlusion. Absorptive dressings are best used for moderate to heavy draining wounds. They are multilayered that include a semiadherent or a nonadherent layer combined with highly absorptive layers of fiber. These fibers are most often consistent with cellulose, cotton, or rayon that effectively manages exudate. Some absorbent dressings form a gel when they come in contact with wound exudate. Absorptive dressings can be used as a primary or secondary dressing. Their main function is to minimize adherence to the wound bed and manage the exudate.

Practice Essentials

- Specially designed to minimize adherence to the wound and effectively manage exudate.
- Initial monitoring may be warranted to assess dressing change frequency to prevent maceration.

Advanced Therapies

Advanced therapies (also referred to as advanced or adjunctive interventions or modalities) have been defined as mechanical, biological, or pharmaceutical modalities. These are utilized when standard basic wound care protocols have failed to produce demonstrable improvement in wound healing, as evidenced by poor granulation development and a flattened wound healing trajectory.[32] These advanced interventions are utilized to promote wound healing via multiple mechanisms including stimulating angiogenesis and enhancing tissue perfusion. It is important to understand that advanced therapies are not typically used as initial or first-line strategies but rather are incorporated into the wound care plan using well-defined guidelines and algorithms.

Based on data published by Dr. Peter Sheehan, wounds that do not demonstrate greater than 50% reduction in size after 4 weeks of basic wound care are unlikely to heal.[33] Patients presenting for wound care consultation should receive aggressive basic wound management based on their initial workup and evaluation. Basic wound care typically includes débridement, control of bioburden and infection, off-loading and pressure relief, edema control, and moist wound healing. Patient progress and response are then followed closely. If at 4 weeks, the wound fails to show at least a 50% decrease in wound size, advanced therapies should then be considered. This recommendation has been suggested by Expert Consensus Panels and adopted by multiple Professional Wound Care Societies as well as included in National Coverage Determinations.[34]

The list of advanced modalities is vast in number and is constantly changing based on advances in technology and our understanding of the basic tenants of the physiology of healing and wound care. The following sections are not intended to be complete or exclusive but rather provide a representative overview of examples of several therapies in each category. The listing of an advanced therapy in one category or another is provided only within a suggested organizational framework for educational purposes.

Mechanical

A large number of devices and therapies can be considered in the group of mechanical modalities. Included in this category are NPWT, compression therapy, and energy-based therapy (electrical stimulation [ES], light, and ultrasonic). Mechanical therapies impact wound healing via several mechanisms including angiogenesis, cellular stimulation, bioburden reduction, and enhanced tissue perfusion.[35]

Negative Pressure Wound Therapy

NPWT is in its third decade of use in the United States and has become an accepted therapy for many chronic and, in some cases, acute wounds. It is commonly used to assist with wound closure. In general, NPWT is indicated for full-thickness wounds that require contraction and granulation tissue formation. Some NPWT systems are appropriate for tunnels, tracts, and undermining (Table 9-5). Reduction of wound volume is one of the most widely recognized outcomes from using NPWT. The National Pressure Injury Advisory Panel (NPIAP), European Pressure Ulcer Advisory Panel (EPUAP), and Pan Pacific Pressure Injury Alliance (PPPI) recommend the use of this modality for the treatment of deep, category/stage 3 and 4 pressure ulcers.[36]

Table 9-5 Indications, Contraindications, and Precautions for Using NPWT

Indications

- Diabetic foot ulcers
- Pressure ulcers (stages 3 and 4)
- Venous insufficiency ulcers
- Arterial insufficiency ulcers
- Full-thickness burns
- Surgical wounds (especially infected sternal wounds)
- Postoperative and dehisced surgical wounds
- Traumatic wounds
- Explored fistulas
- Skin grafts and flaps

Contraindications

- Exposed vital organs (treatment may continue after the organ has been covered by protective mesh)
- Inadequately débrided wounds (granulation tissue that will not form over necrotic tissue)
- Untreated osteomyelitis or sepsis within the vicinity of the wound
- Presence of untreated coagulopathy
- Necrotic tissue with eschar present
- Malignancy in the wound (negative pressure therapy may lead to cellular proliferation)
- Allergy to any component required for the procedure

Special Considerations before using NPWT

- High risk for bleeding and hemorrhage (i.e., anticoagulation therapy, platelet aggregation inhibitors, bleeding disorders)
- Patients with:
  - friable vessels and infected blood vessels
  - vascular anastomosis
  - infected wounds
  - osteomyelitis
  - exposed organs, vessels, nerves, tendon, and ligaments
  - sharp edges in the wound (i.e., bone fragments)
  - spinal cord injury (stimulation of sympathetic nervous system)
  - enteric fistulas
- Patients requiring:
  - MRI
  - hyperbaric chamber (electrical component may not be taken inside the chamber)
  - defibrillation
- Patient size and weight
- Use near vagus nerve (bradycardia)
- Circumferential dressing application
- Mode of therapy—intermittent versus continuous negative pressure

From Sheehan, P., et al. Percent Change in Wound Area of Diabetic Foot Ulcers Over a 4-Week Period Is a Robust Predictor of Complete Healing in a 12-Week Prospective Trial. *Diabetes Care* 26:1879-82, 2003; Snyder, R.J. Consensus Recommendations on Advancing the Standard of Care for Treating Neuropathic Foot Ulcers in Patients with Diabetes. *Ostomy/Wound Management* 56(4 Suppl):S1-24, April 2010. Available at: http://www.cms.gov/medicare-coverage-database/details/ncd-hyperbaricoxygentherapy. Accessed January 24, 2020.

NPWT applies subatmospheric pressure, or suction, to the wound bed by way of a device that is attached to a wound contact layer (interface dressing) through a plastic tube (Fig. 9-11). Several types of interfaces

are used with these devices, including foam, moistened cotton gauze, and nonwoven polyester layers joined by a silicone elastomer.[37,38] Most clinicians have migrated to the use of foam interfaces based on adverse outcomes that have been reported with gauze.[39] The interface dressings are covered with a transparent film or thin hydrocolloid, depending on the device used, that seals the wound and dressing to maintain the vacuum effect. Interface dressings containing silver or other antibiotics are available from some manufacturers. Manufacturers recommend initially changing the dressing 48 hours after beginning treatment and then 2 to 3 times per week as indicated by the wound's response to the NPWT intervention.[37,38,40–44]

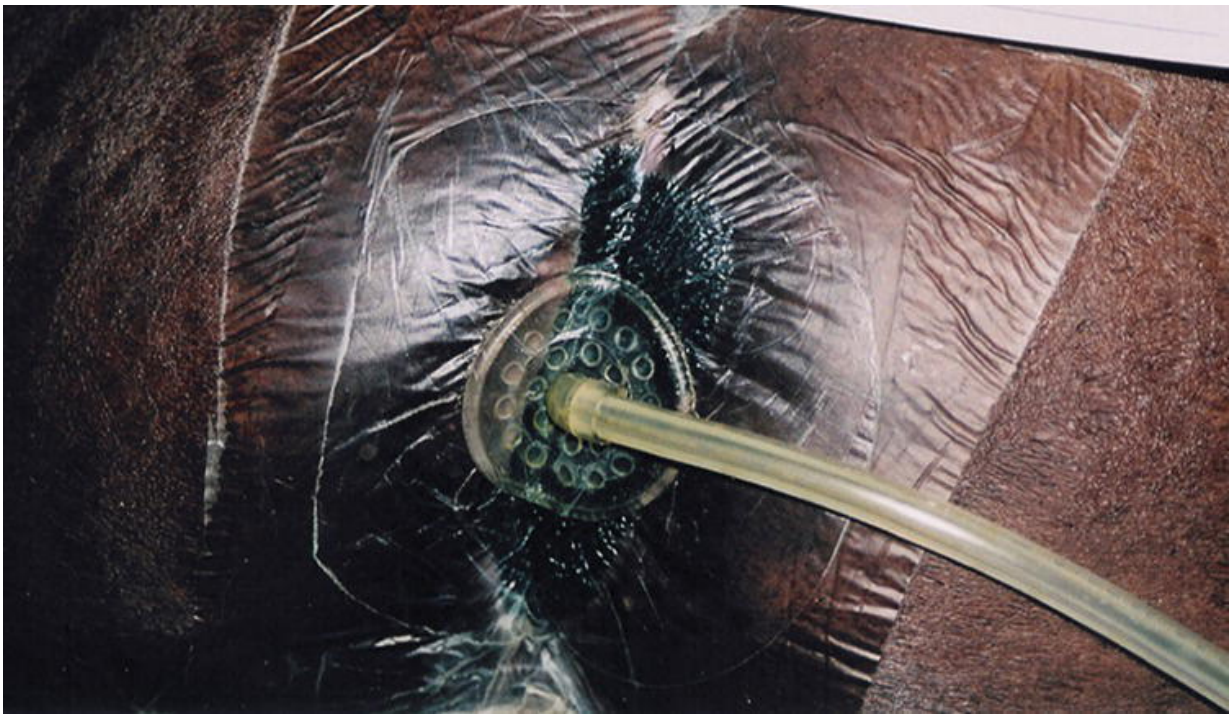

Figure 9-11. Negative pressure wound therapy. (Photos courtesy of S. Baranoski, A. McIntosh, and L. Montoya.)

There are several types of NPWT devices: (a) electrical or battery powered and (b) mechanically powered. The electrical devices were the first type launched and adapted into clinical practice and deliver negative pressure via an electrically or battery-powered pump. Once the wound is cleaned and débrided, the dressing is applied. An evacuation tube runs from the wound through the interface dressing, drawing excess exudate away from the wound and into a canister attached to the other end of the tubing. The canister is attached to the vacuum pump providing either continuous or intermittent negative pressure, adjusted for the type of wound being treated. Negative pressure created by the pump is in the range of 0 to 200 mm Hg

depending on the system used.[40–42] A mechanically powered NPWT device was recently developed using a coiled spring to provide the energy to create the negative pressure. This device has a cartridge providing two functions: (a) contains the spring, which generates continuous negative pressure, and (b) stores fluids removed from the wound (Fig. 9-12).

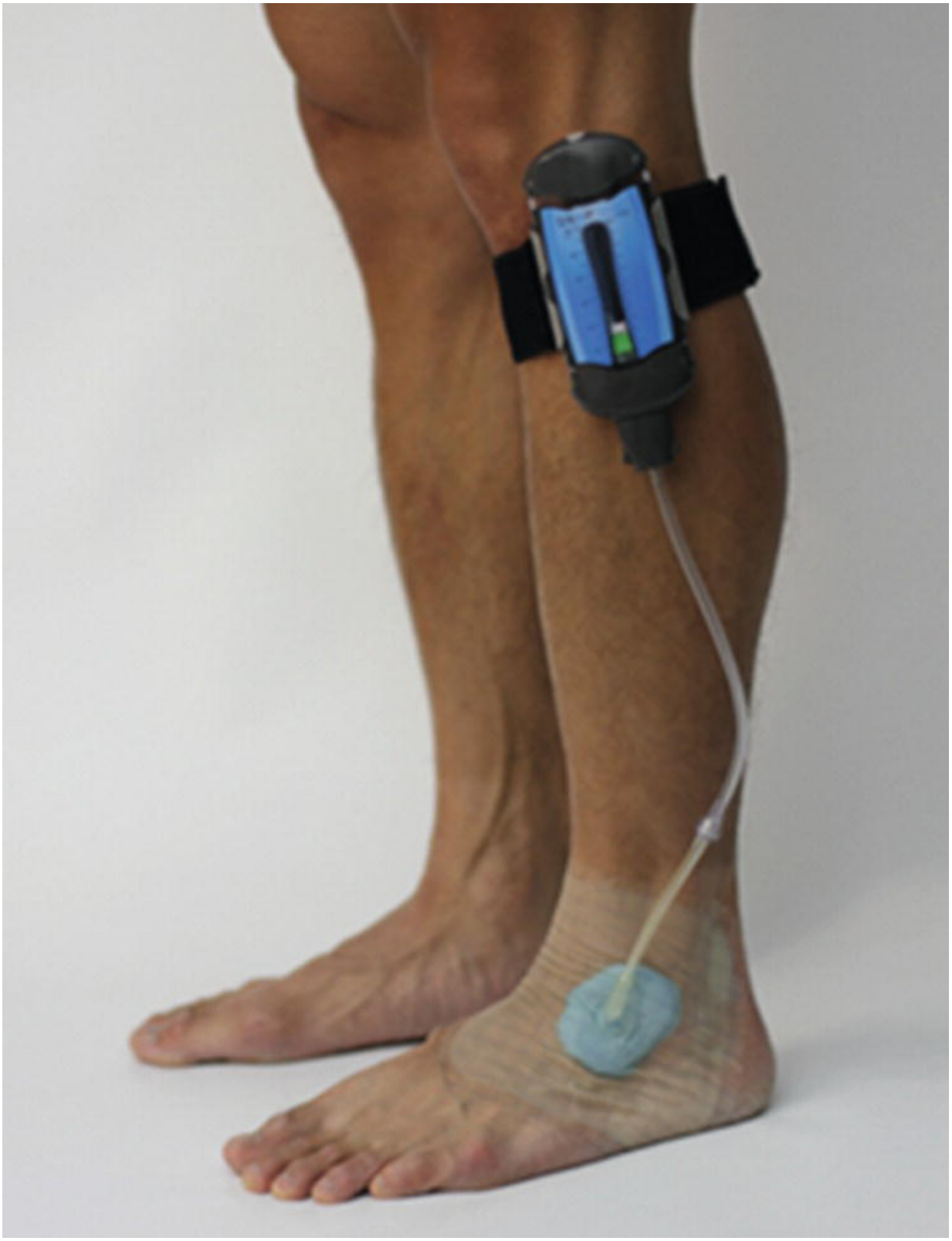

Figure 9-12. SNaP unit on a mobile highly functional patient. (Courtesy Spiracur Inc.)

Comparison studies between the two types of units, electrically powered and mechanical (coiled spring) powered, have been performed in clinical practice. The results of these studies demonstrate no wound closure advantage or disadvantage of one type of power delivery system over the other.[45–47] In general, deciding which unit to use will be determined by the size of the wound, exudate amounts, and patient convenience and comfort. For instance, large wounds with high exudate levels will need the larger canisters found with electrically powered units, whereas smaller wounds with little exudate, especially in very mobile patients leading highly functional lives, may be appropriate for the mechanically coiled spring unit.

The application and type of dressing vary by the specific manufacturer and the goals for use of the device. NPWT reportedly facilitates wound closure and healing through several mechanisms of action, including:[36–38,40–42]

- facilitating wound contraction/retraction
- removing edema, which improves nutrient and oxygen delivery
- removing wound exudate, which may be a medium for bacterial colonization.
- decreasing harmful levels of proinflammatory agents, such as MMPs, found in chronic wounds
- promoting angiogenesis.

One theory as to how NPWT enhances wound closure is that the externally applied stress creates macrostrain and microstrain in the wound bed and in individual cells.[48–51] Macrostrain is the physical response that can be seen immediately as the negative pressure contracts the wound.[48] In addition, this mechanical stress creates changes at the cells' surface (also known as microdeformations), causing growth factors and cytokines to "up-regulate" fibroblastic activity, increasing production of the ECM and cell proliferation within the wound and ultimately creating new granulation tissue.[48–51] This

increase in the rate of granulation tissue formation has been noted in several studies using NPWT.[48–51]

According to the NPIAP,[36] wound volume was significantly reduced, and healing was more rapid in pressure ulcers treated with NPWT compared with other traditional wound treatments.

It has been suggested that NPWT assists in preparing the wound bed for closure once granulation tissue has been well established.[51] When a wound is filled with newly synthesized granulation tissue, resurfacing by epithelial cells can take place more readily.[51]

It is important that the wound be débrided of as much necrotic tissue as possible before using the NPWT device. The exact percentage of necrotic tissue that can be present in the wound bed varies according to manufacturer as well as facility and clinician protocols. In general, it is best to have the wound bed *free* of necrotic tissue prior to application of the NPWT device.

Many clinicians are now combining NPWT with the instillation of infusion of solutions into the wound interface. A variety of solutions have been reported and include normal saline, hypochlorous acid, and other antimicrobials, which serve to enhance wound irrigation and cleansing as well as decrease bacterial colonization across the surface of the wound. Some manufacturers have provided devices that have been modified to allow this technique, while some clinicians report excellent outcomes with inexpensive simple dressing modifications that they customize themselves. Patients can easily manage this independently in the outpatient setting using the Modified Intermittent Therapeutic Infusion Technique[52,53] (Fig. 9-13). Complication with excessive bleeding can occur in individuals with blood vessel grafts in the leg and breastbone and groin wounds, patients receiving medication for blood clots, and during wound dressing removal that was attached to the tissues (Fig. 9-14).

Figure 9-13. Modified Intermittent Therapeutic Infusion (MITI NPWT Technique). (Photo courtesy of J.A. Niezgoda.)

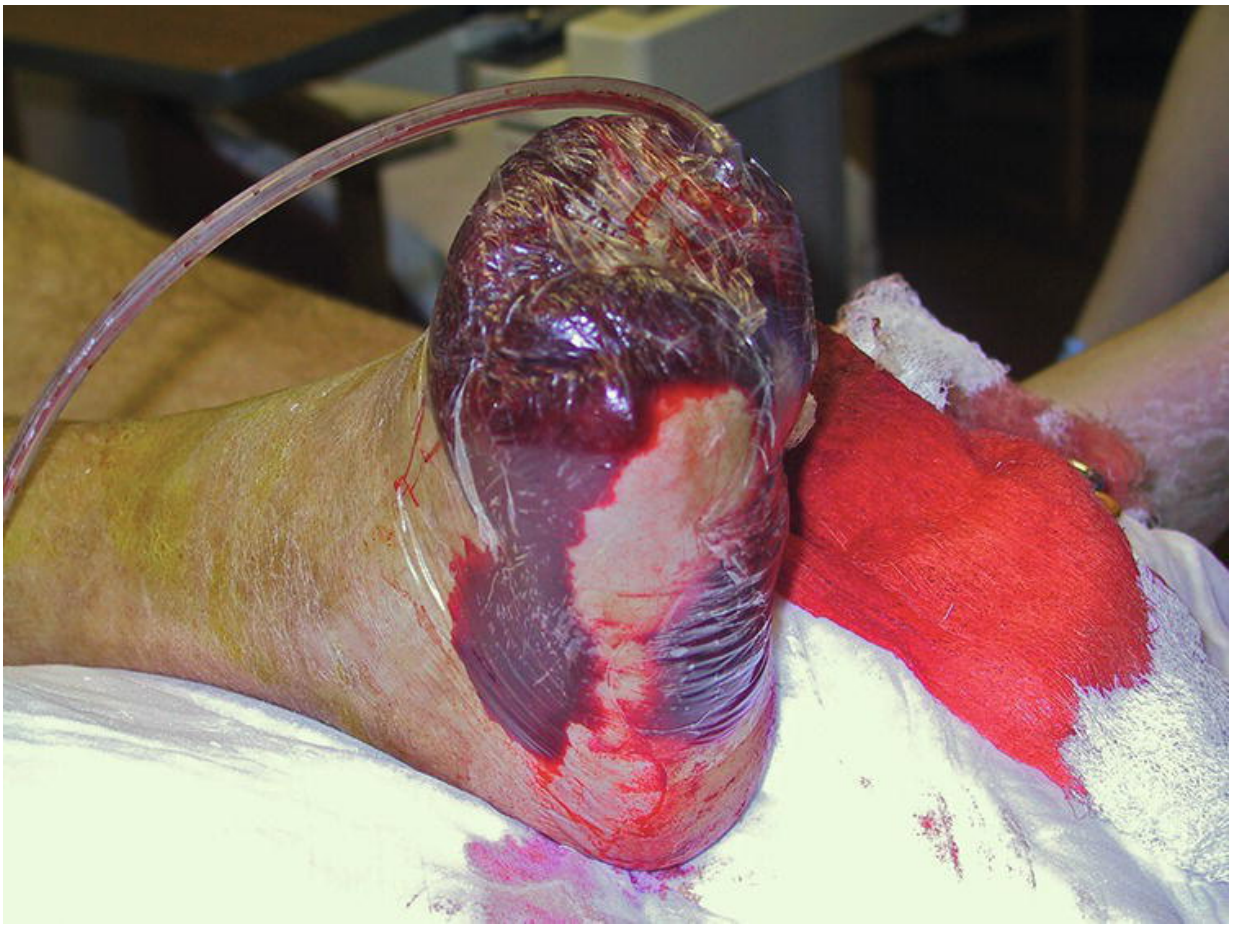

Figure 9-14. NPWT complication from bleeding. (Photo courtesy of J.A. Niezgoda.)

Practice Point

NPWT should be used cautiously when there is active bleeding, when the patient is on anticoagulants, when there is difficult wound hemostasis, or when placing the dressing in proximity to blood vessels.

Practice Essentials

- Device operation and utilization should be performed according to the specific manufacturer’s directions.
- NPWT is not a substitute for wound débridement, and wounds treated with NPWT should be well débrided prior to initiation of NPWT.
- Application of skin prep or sealant to the periwound area may be helpful in preventing blistering, denuding, and excoriation when removing the adhesive drape.
- Make sure all dressing materials (gauze or sponges) are removed from the wound bed during NPWT dressing changes.

Compression Therapy

Venous Compression Therapy

Compression therapy is the foundation for successful management in patients with edematous wounds caused by venous insufficiency and/or lymphedema. Compression therapy wraps are used to manage fluid accumulation and promote sufficient return of venous blood back to the central system and lymphatic fluid back into the bloodstream and lymphatic system. The substances transported by the lymphatic system are called *lymphatic loads* and consist of protein, water, cellular debris, and fat from the digestive system. These loads are filtered by the regional and central lymph nodes prior to re-entry into the venous and lymphatic systems.[54]

It is worth noting that the edema associated with venous insufficiency is different in consistency from the edema or fluid accumulation that is seen in lymphedema. Protein-rich lymphedema fluid appears to be more “viscous” or thicker than the edema associated with venous insufficiency and requires different treatment interventions, including higher inelastic or short-stretch compression forces when using wraps or garments.[55] (See Chapter 15, Venous Disease.)

Compression Wraps

Several types of compression wraps are available, and instructions on their application techniques vary. Some have layers that are applied in a spiral fashion, while others are wrapped in a figure-eight configuration, and still others use a combination of the two wrapping techniques. Bandages are also classified by their elasticity. Short-stretch bandages have a smaller degree of stretch than do long-stretch bandages. They are applied in layers allowing higher compression. Short-stretch or rigid compression systems are particularly suited to managing the fluid accumulation of lymphedema and are also indicated for patients with a combination of venous insufficiency and lymphedema in the same leg.[56] An Unna boot is an inelastic system that includes a moist layer impregnated with several substances, including zinc oxide, calamine, and gelatin. This layer molds to the extremity, becoming semirigid when dried. Both inelastic

and short-stretch systems create high working pressures during ambulation and low resting pressures during rest; this improves the calf pump function to facilitate fluid return to the central system and decreases edema in the afflicted extremity.

Practice Point

When using an Unna boot, apply a nonadherent dressing over the open wound first so as to avoid damage to the healing tissue when removing the compression dressing.

Long-stretch bandages are so called due to their large amount of extensibility and elastic recoil back to near-original configuration. While short-stretch bandages require the patient to be ambulatory or able to engage the calf muscles effectively (ideally by walking), long-stretch bandages have been shown to be suitable for individuals who are not active or who are nonambulatory.[56] However, due to the high working pressure and low resting pressure associated with inelastic or short-stretch devices, there is evidence to suggest that the reduction in venous reflux may be more effective than with elastic devices, for reducing ambulatory venous hypertension and edema.[57]

While 78% of nurses who responded to a survey indicated that compression wrapping/bandaging multilayer system is the gold standard for treating venous ulcers, only 68% indicated that they know how to apply these compression wraps.[58] Clinicians need to be trained and skilled to proficiently and safely apply these compression wraps, keeping in mind that they should be applied according to the manufacturer's detailed directions[59] (Fig. 9-15).

A

B

C

D

Figure 9-15. Application of a multilayer compression system. **(A)** Absorbent padding applied to the leg. **(B)** Application of a light conformable dressing applied in a spiral manner. **(C)** Flexible cohesive bandage applied over the two other layers, also in a spiral manner. **(D)** Completed compression wrap.

Compression Devices

Some compression systems do not fall into the wrap or bandage category. These include garments that are usually short-stretch systems consisting of material that does not give way during ambulation; these systems often include Velcro straps to help secure and conform the garment to the extremity contours (Fig. 9-16). Application techniques vary according to product; it is important that the clinician read the package insert before applying the compression system. There are compression sock systems that include a liner that goes over the wound/ulcer dressings and an over sock that provides the recommended level of compression.

A

Figure 9-16. Short-stretch bandages, such as the CircAid **(A)** and CompreFit BK **(B)**, do not give way when the calf muscle pump is exercised. The force of

the muscle is directed back into the leg and promotes venous return.

Intermittent Pneumatic Compression

Intermittent compression therapy or external pneumatic compression therapy contributes to the healing of venous insufficiency ulcers by collapsing the superficial venous system and forcing blood into the deep system, thus increasing subcutaneous pressure. Intermittent pneumatic compression (IPC) prevents the leakage of blood, fibrin, and protein from the skin capillaries. In addition to the hemodynamic effect of IPC, enhancement of fibrinolysis has been an important outcome of this intervention.[60–62] IPC results in improved circulation with an increase in oxygenated blood flow and the removal of potentially harmful toxic waste from the wound and periwound area.[62]

Application of IPC to the lower extremity is best accomplished with the patient positioned supine and the extremity elevated above the heart. Treatment time varies but usually lasts from 45 to 60 minutes.

IPC is contraindicated for patients with acute deep vein thrombosis or severe peripheral vascular disease. Although not an absolute contraindication, vigilant observation and communication with the patient who has a history of congestive heart failure must be practiced when using any type of compression system. This is important in order to ensure that the heart is not overloaded with the increased fluid return back to the central system.[62] There is some controversy regarding the use of IPC in patients with lymphedema due to concerns for damaging the superficially located lymphatic anatomical structures, due to high pressures.[63]

Practice Point

- Compression therapy should be applied cautiously and at lower levels of compression (20 mm Hg) when there is evidence of mild to moderate peripheral arterial disease.
- If a patient presents with mixed etiology of venous and arterial insufficiency, he or she should be monitored diligently to ensure that arterial compromise is not occurring.

Vascular assessment and an ankle–brachial index Doppler study are critically important prior to the use of compression therapy dressings or devices. (See Chapter 15 “Venous Disease” and Chapter 16 “Lymphedema Management.”)

Arterial Compression Therapy

Compression therapy is generally associated with venous insufficiency; however, compressive devices have also been successfully utilized for patients with arterial disease. Pneumatic compression therapy modalities are external limb devices that apply an end-diastolic compressive force to the involved extremity, which enhances distal blood flow and tissue perfusion (Fig. 9-17). These devices also improve venous return and tissue edema, which in turn enhances tissue perfusion by decreasing the extrinsic compression of the arterial vasculature by edema-associated pressure. Multiple studies have demonstrated symptom improvement and enhanced wound healing via vascular augmentation as evidenced by improved transcutaneous oxygen measurements, ankle–brachial indices, toe pressure, and pulse volume recordings. Intermittent compression[64] has profound effects on the release of anti-inflammatory, vasoactive, and anticoagulatory mediators and substances. Arterial pneumatic compression has gained acceptance for patients with end-stage arterial vascular disease or for those with failed attempts at vascular reconstruction.[65,66] Improved clinical outcomes have also been reported when using arterial compression therapy in combination with hyperbaric oxygen therapy (HBOT).[67]

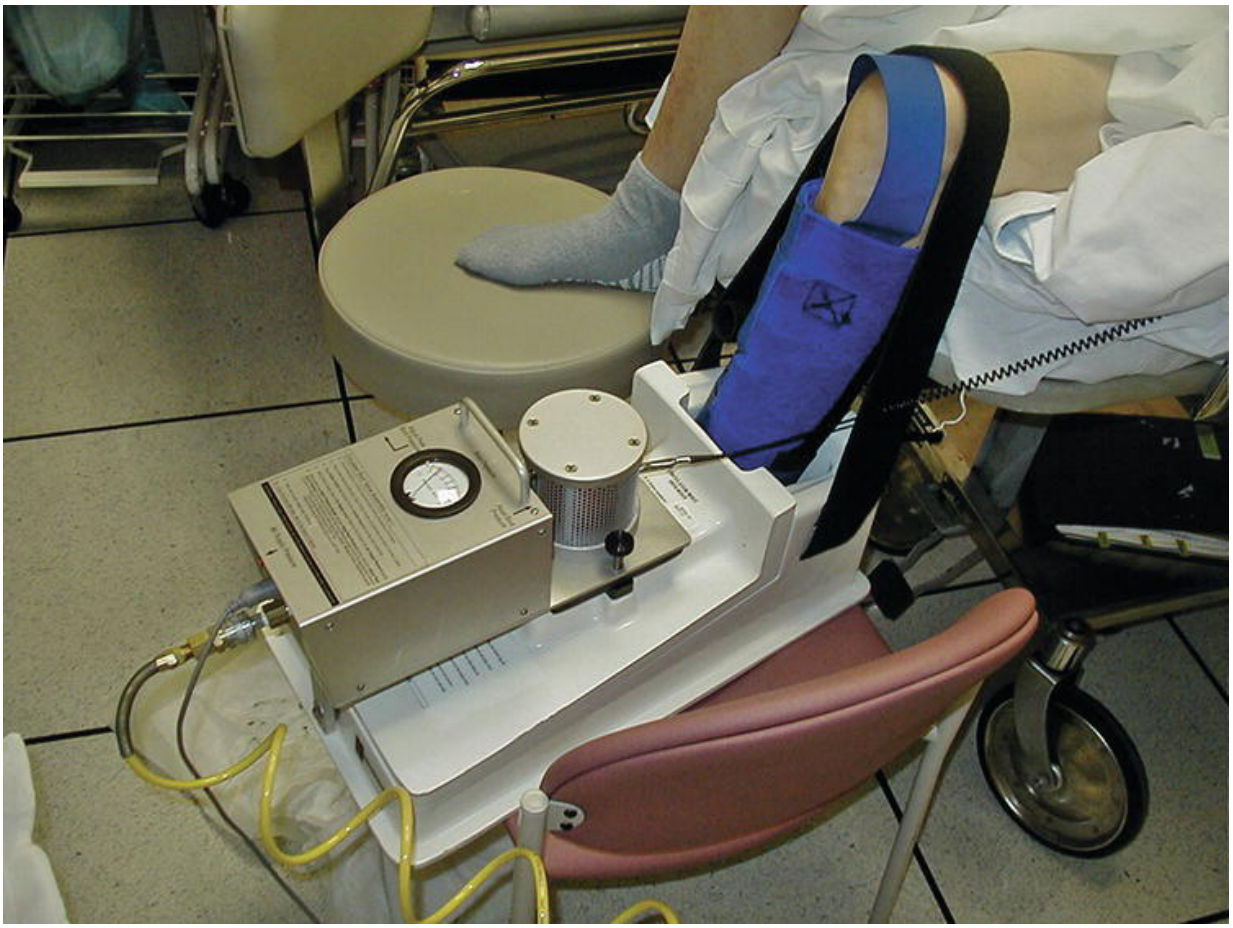

Figure 9-17. Arterial assist compression device. (Photo courtesy of J.A. Niezgoda.)

Energy-Based Therapy

This section discusses various types of energy-based technologies and the application of these devices when used to enhance wound closure and healing. All energy-based therapies should be used in conjunction with appropriate moist wound healing practices according to the advanced therapy algorithm previously discussed. The application of these exogenous energies has been shown to augment cellular, tissue, and vascular responses in both acute and chronic wounds.[36]

Practice Point

- NPIAP Statement: Biophysical (energy-based therapy) agents should be directed by and under the supervision/management of a skilled licensed professional educated and trained in safe and effective selection, application, and monitoring methods for these devices.[36]

Clinicians are advised to refer to Local Coverage Determinations regarding level of evidence and reimbursement issues related to individual energy-based therapies discussed in this section.

Electrical Stimulation

In the 18th and 19th centuries, when Galvani[68,69] and Moruzzi[70] made preparations of cut sciatic nerve and muscle tissue from an excised frog leg, they found that small electrical currents were generated at the cut points. The electric potentials associated with these cut points are now referred to as *injury potentials*. In 1860, German physiologist Emil du Bois-Reymond[71] measured a direct current flow out of a cut in his finger and observed the disappearance of these currents when the wounds healed. This theory of this physiological response to injured skin is termed the “current of injury.” The reason this is important in chronic wound closure and healing is that ES can facilitate “restarting” the stalled wound healing sequela of events.

ES has been used for more than three decades to accelerate the rate of chronic wound healing. This energy has been given strong evidence of as an adjunctive therapy for treatment of recalcitrant stage 3 and 4 pressure injuries.[36] Healthcare professionals who have used ES consider it to be one of the most cost-effective, therapeutically efficacious tissue repair and wound healing accelerators in our wound care toolkit.

Practice Point

Electrical stimulation facilitates “restarting” chronic wounds that have stalled in the healing processes, regardless of the phase of healing.

During the inflammatory phase of healing, ES has been shown to increase blood flow and skin temperature. ES induces a faster inflammatory response by increasing vascular vasodilation and therefore tissue oxygenation. In the proliferative phase of wound healing, ES demonstrated the ability to increase angiogenesis, collagen matrix formation, and wound contraction and epithelialization. During the last phase of healing, the remodeling phase, ES has shown to produce an advanced remodeling by increasing cellular activity.[72]

Unfortunately, ES is not widely used due to nonstandardized clinical algorithms combined with the lack of knowledge, education, and training in the application of this energy.[73] In 2014, the Paralyzed Veterans of America published a Clinical Practice Guideline titled *Pressure Ulcer Prevention and Treatment Following Spinal Cord Injury,*[74] which stated that ES qualified as a “stand-alone intervention” and should no longer be classified as an adjunctive therapy. The reported effects of ES in relation to chronic wound healing and tissue repair include increased blood flow,[75] increased tissue oxygenation,[76,77] increased fibroblast proliferation and collagen deposition,[71] increased angiogenesis,[78] decreased wound pain,[79] increased wound tensile strength,[80,81] and decreased diabetic peripheral neuropathic pain.

ES energy may be considered for treatment of lower limbs with severely compromised arterial blood flow. ES energy delivered as high-voltage pulsed current (HVPC) applied to ischemic lower

extremities has been shown to increase transcutaneous pulse oximetry and perfusion, with subsequent healing of these legs and feet in patients who were at high risk for amputation.[71]

The application of ES in wound healing includes therapeutic exogenous (externally applied) electrical currents. These currents are delivered to the wound tissues by using at least two electrodes, one of which may be applied directly into or both around the wound (periwound tissue), or by applying a stocking or glove electrode garment to the affected limb (Fig. 9-18).

Figure 9-18. Electrical stimulation with use of a stocking electrode garment. (Photo courtesy of P. Scarborough.)

The electrical current may be delivered as low-intensity direct current, HVPC, transcutaneous electrical nerve stimulation, or pulsed electromagnetic energy. However, HVPC has become the current used most often for ES wound treatments in the past decade.

ES is performed by physical therapists or other licensed healthcare professionals who are educated and trained in identifying how and when to apply and change the ES treatment parameters (e.g., ES dosage, polarity, electrode placement) according to the wound characteristics. According to Myer,[82] HVPC has a waveform of paired short-duration pulses with a long interpulse interval. It's a pulsed or interrupted monophasic waveform. The duration of treatment is usually 45 to 60 minutes, delivered 5 to 7 times per week. ES is indicated for all types of wounds regardless of the etiology. It is contraindicated for basal or squamous cell carcinoma in the wound or periwound tissue, for osteomyelitis (if the patient is not responding to systemic treatment with antibiotics), for ion residues of iodine or silver in the wound, and over electronic implants or directly over the carotid arteries or heart.[73]

The decision-making process for how to use ES in a specific wound depends on the phase of wound healing and what the clinician is attempting to accomplish. The effect of ES on tissues has been widely studied in many cell types in chronic wounds.[83] Many cell types show directional migration toward an external electric field. These cell types include epithelial, fibroblasts, lymphocytes, macrophages, endothelial neuronal cells, and stem cells.84 This process is known as galvanotaxis or electrotaxis.

ES delivers a summation of either a positive charge or a negative charge to the tissues. Therefore, it is important to consider what specific outcome the clinician is attempting to create. For instance, if treating an infection is important, the current selected would be a summation of a positive charge to attract the negatively charged neutrophils, whereas if the goal is to facilitate the proliferative phase for angiogenesis and granulation tissue formation, one would want a summation of the negative current to attract the positively charged fibroblast. However, in a recent randomized, controlled clinical study, Polak and colleagues showed that overall ES improved blood flow and

wound area reduction rate whether it was applied with either a positive or a negative charge to the wounded tissues.[85]

Practice Point

- Don't place ES electrodes over the carotid sinus, close to the heart, or near the laryngeal musculature.
- When using silver products, ensure that the residue has been well irrigated from the wound prior to using ES.

Light Therapy

Light is a form of electromagnetic radiation (EMR). Sunlight has been known for many centuries to have beneficial effect on humans. The Egyptians, Greeks, and Chinese all have writings referring to the use of sunlight to treat different conditions and illnesses. Sunlight is still prescribed for certain illnesses today. Most of the time, however, light therapy, or phototherapy as it is known in the healthcare field, involves the application of specific wavelengths to patients using various medical devices.[86]

Light comes in different colors, spreading across the rainbow of hues known as the visible spectrum. Each color in this spectrum corresponds to a different wavelength. The visible light spectrum ranges from short wavelengths of the violet or purple color to long wavelengths, which are red. Photons of light from the violet end of the spectrum have the highest energies and the highest frequencies, while red photons have lower energies and lower frequencies. Beyond the range of our human vision are the longer wavelengths of the infrared and the shorter wavelengths of the ultraviolet regions of the electromagnetic spectrum (EMS).

Low-Level Laser Therapy

When discussing phototherapy in wound care today, one is usually referring to a low-level or cold laser, which is used for tissue regeneration, as opposed to coherent laser beams of high-intensity light, which is used for surgical incisions. While laser therapy for tissue regeneration is still in its infancy in the United States, it has been used in Europe and Russia for more than 30 years as an adjunctive modality for wound healing. “Laser” is an acronym for light amplification by stimulating emissions of radiation. Low-level laser therapy (LLLT) or low-intensity laser therapy (LILT) is also known as *cold laser therapy*, *photobiomodulation*, or *monochromatic infrared light therapy.* Laser light is always one single color (therefore monochromatic) and is in the infrared (nonvisible) area of the light spectrum. Several studies have demonstrated a positive effect of LLLT on the three overlapping phases of wound healing: inflammation, proliferation, and remodeling. The proposed outcome of LLLT is more rapid wound healing. Dyson and colleagues[87–94] have suggested that this modality can reduce the inflammatory phase, cause earlier initiation of the proliferative phase, and augment the rate of contraction as angiogenesis increases. More than 2,000 scientific, peer-reviewed studies on the uses of LLLT have been published worldwide. Specific topics have included chronic wound healing, acute soft tissue injuries, shingles, regeneration for both nerve injuries and diabetic peripheral neuropathy, and reducing postoperative pain. There are also a significant number of research articles describing the effects of LLLT on keratinocytes, mast cells, and macrophages and fibroblasts, all of which are critical to the wound healing processes. Kazemikhoo and colleagues[95] reported that the use of LLLT significantly decreased the rate of dehiscence in patients with grade 3 burn ulcers in a randomized clinical trial. Taradaj and colleagues[96] showed that effective healing of pressure injuries is seen in ulcers treated with laser irradiation at a wavelength of 658 nm.

Light is applied by a multicluster diode probe (Fig. 9-19), a single diode probe, or a multicluster diode pad. The literature and anecdotal reports indicate that most chronic wounds, including diabetic neuropathic foot ulcers, venous insufficiency, arterial insufficiency, and pressure ulcers, respond well to this treatment intervention.[97–107]

Figure 9-19. Multicluster diode probe for use in low-level laser therapy for wound healing. (Photo courtesy of P. Scarborough.)

Practice Point

When treating with LLLT directly over or in an open wound, apply a thin plastic transparent barrier over the wound or place the pad(s) or probe in a plastic bag to keep them free of contaminants.

Ultraviolet Light

Ultraviolet light (UV) has been used for centuries to treat a myriad of health and skin problems in the form of natural sunlight and more recently by artificial UV-generated sources. Ultraviolet light A (UVA) (wavelength 315 to 400 nm) and ultraviolet light B (UVB) (wavelength

280 to 315 nm) are responsible for the pigmentation and erythema (and sometimes blistering) often seen in light-skinned individuals after significant sun exposure. UVB light assists in wound healing by inducing an inflammatory reaction, stimulating the growth of granulation tissue, and promoting breakdown and elimination of dead tissue from the wound.[108]

Ultraviolet light C (UVC) (wavelength 200 to 290 nm) is the form of UV light most often used in the treatment of chronic wounds specifically for its bactericidal effects. Recent research demonstrated that UVC is capable of killing strains of bacteria in laboratory cultures, in animal tissue, and in patients with chronic ulcers infected with methicillin-resistant *Staphylococcus aureus.*[109,110] In patients with chronic wounds, UVC treatment also reduced the wound bioburden and facilitated wound healing.[111] In addition, in vitro studies have shown that UVC therapy destroyed 100% of antibiotic-resistant bacteria.[112] However, UVC and UVB have shown damage to host cells; therefore, the clinician must balance the benefit the ulcers healing.[113]

Practice Point

Contemporary UV light equipment is usually equipped with distance guards, which ensure correct distance and reproducibility of UV treatments.

Ultrasonic Energy

Therapeutic ultrasound delivers energy through mechanical vibrations in the form of sound waves at frequencies above detection by the human ear (>20 kHz). Ultrasound affects tissue through thermal and nonthermal mechanisms, which are determined by the physical properties, which are associated with ultrasound: (a) frequency or the number of oscillations a molecule undergoes in 1 second and (b) intensity or level of power. The thermal effects associated with greater tissue absorption of energy increase with higher frequencies, whereas nonthermal effects are predominant with lower frequencies and when ultrasound is pulsed.[114] Ultrasound is nonionizing radiation and therefore does not impose the hazards attributed to ionizing radiation such as cancer production and chromosome breakage.[115]

High-Frequency Ultrasound

High-frequency ultrasound is currently used in the range of 1 to 10 MHz primarily for diagnostic fetal imaging and vascular duplex scanning. High frequencies of ultrasound have shorter wavelengths and are absorbed more easily.[116]

It has also been used in the 1 to 3 MHz range to promote soft tissue injury healing and occasionally reported to facilitate wound healing. High-frequency ultrasound devices create their effect by running electricity through a crystal in the sound head, causing the crystal to “vibrate.” The vibrations are then passed through the sound head via an ultrasound coupling medium into the tissues, causing them to vibrate and creating a local thermal effect.

Low-Frequency Ultrasound

Recently, low-frequency ultrasound (LFU) has been added to the arsenal of tools available for wound care and is the most common type of US device used in wound care today. Low frequencies penetrate deeper in tissue and are typically transmitted at frequencies of 20 to 120 kHz.[116]

Delivery of LFU to wounds has been shown to effectively débride necrotic tissue, eradicate some strains of bacteria from the wound, and facilitate the wound healing process.[117]

LFU therapy is believed to promote wound healing via two mechanisms: (a) cavitation (production and vibration of micron-sized bubbles within the coupling medium and fluids within the tissues) and (b) acoustic streaming (movement of fluids along acoustic boundaries). The combination of cavitation and acoustic streaming, both of which occur more frequently with kilohertz than megahertz ultrasound, provides a mechanical energy capable of altering cell membrane activity and, therefore, cellular activity.[118] Cavitation is the vibrational effect of ultrasound on micron-sized gas bubbles that form in the blood and lymph and tissue fluids and in the ultrasound coupling media solutions. Periods of high and low pressure in treated tissue can cause the bubbles to increase and decrease in size. Cavitation can be both stable and unstable. Stable cavitation acts to enhance the acoustic streaming phenomena. Unstable or transient cavitation occurs when the microbubbles significantly increase in size and violently implode during the low pressure part of the US wave cycle. On implosion, the bubbles release a large amount of energy, which is destructive to bacteria while being essentially harmless to healthy viable tissue and cells. The tiny shock waves produced by bubble implosions cause preferential and rapid liquefaction and fragmentation of adherent necrotic fibrin (fibrinolysis) and the destruction of microorganisms on wound surfaces.[119]

Acoustic streaming has been shown to alter cell membrane permeability and second messenger activity, which in turn may result to increased production of growth factors as well as macrophage

activity and fibroblast proliferation and migration creating a collagen-rich connective tissue matrix.[120,121] The fibroblasts then differentiate into myofibroblasts to facilitate the contraction process of wound repair. The mechanical energy from an ultrasound wave is absorbed by an individual protein molecule, theoretically inducing a conformational change. Signal transduction pathways can also be stimulated from ultrasound-generated mechanical energy. This may result in a broad range of cellular effects that impact wound healing, including leukocyte adhesion, growth factor and collagen production, increased angiogenesis, increased macrophage responsiveness, increased fibrinolysis, and increased nitric oxide levels.

Contact and Noncontact Technology

There are currently two categories of ultrasonic devices available for the delivery of LFU in wound care. Noncontact devices allow for the treatment of wounds with ultrasonic energy without directly contacting the tissue or causing thermal effects. The energy is transferred to the wound via the coupling solution (normal saline), which has the appearance of a mist over the wound surface. These devices stimulate the tissue with both acoustic streaming and cavitational effects but do not physically débride tissues. Contact devices also transfer ultrasonic energy to the wound but accomplish this with the combined effects of the coupling solution and the mechanical action of the probe tip. The probe oscillates at the programmed device frequency. This vibrational energy allows for sharp débridement of the wound in addition to wound stimulation via cavitation and acoustic streaming. Based on the vibrational energy of the probe tip, heat is generated during the procedure. While the heat is typically dissipated via the coupling solution, the clinician must be aware of the potential for focal heating if the probe tip is not moved continuously across the wound base during débridement. The available devices vary in frequency and other features that distinguish them from operationally (Table 9-6).

Table 9-6 Low-Frequency Ultrasound Summary and Comparison of Specifications

	SonicOne	Sonoca 180	Arobella Qoustic Wound Therapy System	MIST Therapy System	Panacea Qoustic Wound Therapy System
Operational Features					
Frequency	22.5 kHz	20–80 kHz	35 kHz	35 kHz	40 kHz
Intensity	Variable: auto gain control	Variable: 40%–100%	Variable: 10%–100%	Preset	Preset
Mode	Continuous or pulsed	Continuous	Continuous or pulsed	Continuous	Continuous
Coupling	Sterile saline	Sterile saline	Sterile saline	Sterile saline vapor	Oxygenated sterile saline vapor
Controls	Foot pedal	Foot pedal		Button on hand piece	Trigger on hand piece
Treatment time	Usually 2–5 min	Usually 2–5 min	Usually 2–5 min	Usually 5–10 min	Usually 5–10 min
Wound contact with applicator	Yes; autoclavable metal probes	Yes; autoclavable metal probes	Yes; autoclavable curette shaped	No Disposable applicator	No Autoclavable applicator
Clinical Features					
Selective excisional débridement	Yes	Yes	Yes	No	No
Antibacterial	Yes	Yes	Yes	Yes	Yes
Associated pain	Yes	Yes	No	No	No
Aerosolization	Yes	Yes	Yes	Yes	Yes

Adapted and used with permission from Luther Kloth, PT, MS, FAPTA, CWS, FCCWS and Jeffrey Niezgoda, MD, FACHM, MAPWCA, CHWS.

Noncontact Nonthermal Devices

There are two noncontact nonthermal devices. These include the MIST Therapy System (Celleration Inc., Eden Prairie, MN) and the Qoustic Panacea Wound Therapy System Model AS1000 Series (Arobella Medical, LLC, Minneapolis, MN). The MIST device operates at a frequency of 40 kHz in the continuous mode and promotes healing by actively stimulating cells, reducing the bioburden, and increasing blood flow to the immediate treatment area[122] (Fig. 9-20). The Panacea device operates at a frequency of 35 kHz in the continuous mode and provides the same wound healing properties provided by MIST, but with the addition of an oxygenated irrigant. Thus, the Panacea provides the benefit of noncontact nonthermal ultrasound with the additional benefit of topical oxygen.

A

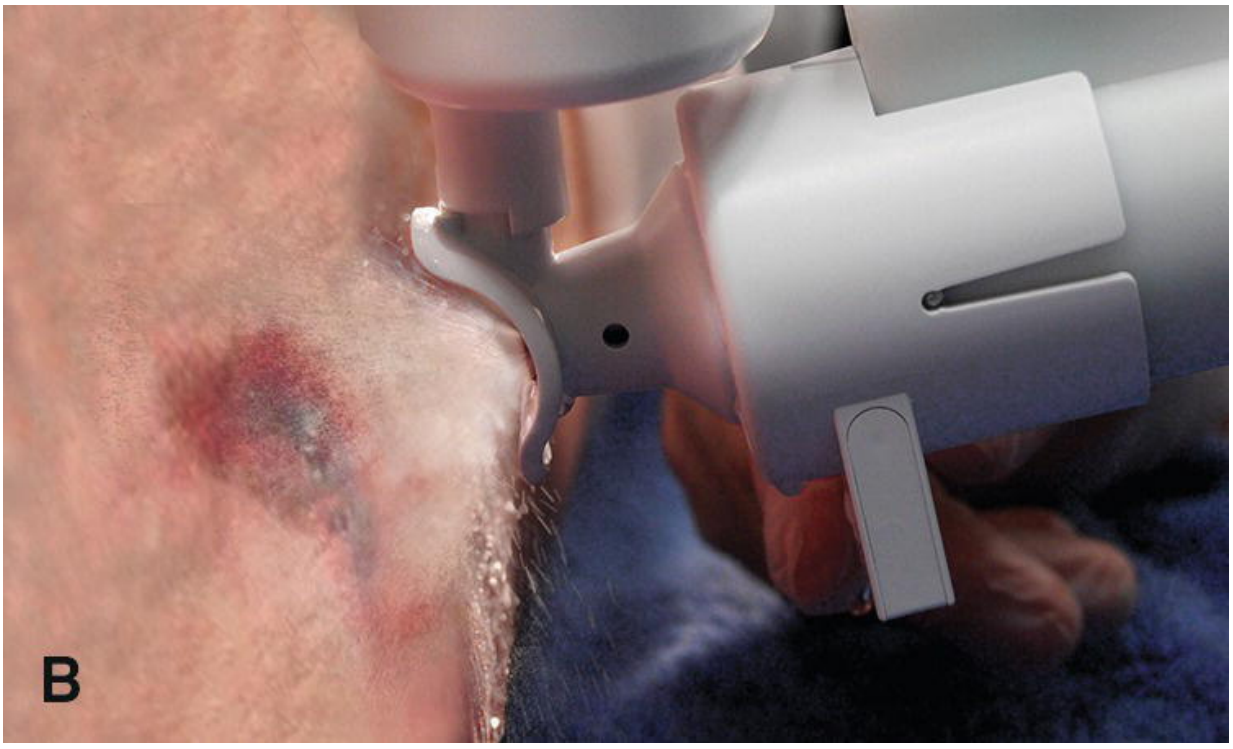

Figure 9-20. (A, B) MIST therapy. (Courtesy Celleration, Inc.)

Contact Thermal Devices

In contrast to the noncontact nonthermal energy transfer devices, the contact thermal devices can accomplish sharp selective and excisional débridement and are often referred to as ultrasound-guided débridement (UGD). It provides selective, precise, and gentle fragmentation of soft and hard tissues, with preservation of healthy tissue through ultrasonic separation of damaged tissue (Fig. 9-21A and B).[123] This cleanses the wound area with less pressure and pain and also improves granulation formation of treated tissue. All UGD devices allow for deep tissue penetration of the ultrasonic energy and cause bacterial cell destruction within the wound bed through combined vibrational and cavitational effects. Continuous irrigation with the coupling solution provides a medium for cavitation and flushes the wound of necrotic tissue fragments and fibrin deposits mobilized during the débridement while preserving the granulation tissue. Various clinical studies have evaluated the safety and efficacy of LFU therapy in patients with a variety of wounds, including recalcitrant pressure ulcers, chronic lower extremity leg and foot ulcers, and diabetic foot ulcers.[118] Studies have suggested noncontact LFU as beneficial in helping to resolve or mitigate the severity of suspected deep tissue injury (DTI) when applied early in the sequelae of tissue destruction from these wounds. More research is needed to determine the full effects and outcomes of noncontact LFU on DTI; however, the questions posed by preliminary studies are creating interest in determining if this biophysical energy can indeed decrease the severity of tissue destruction caused by DTIs.[124]

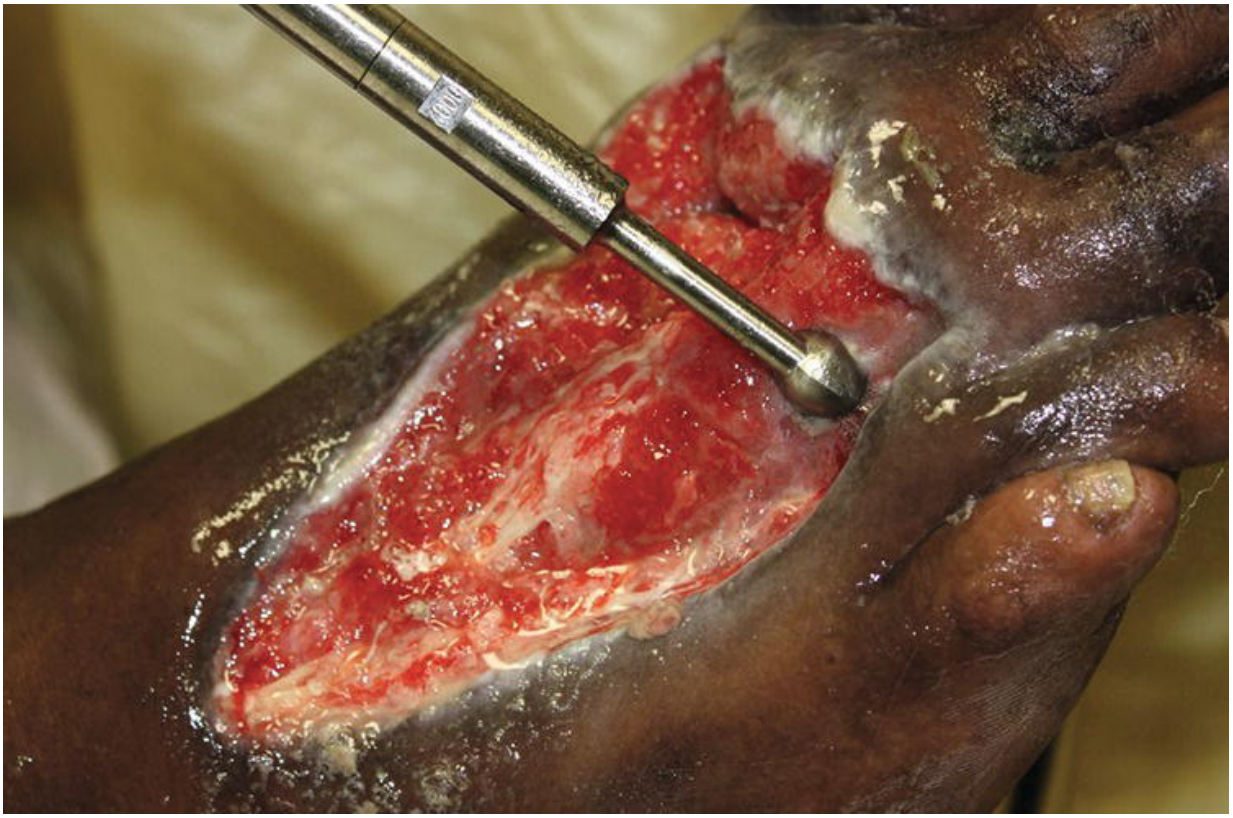

Figure 9-21. Ultrasound-guided débridement (UGD). (Photos courtesy of J.A. Niezgoda.)

Practice Point

- *Ultrasound* cannot be used near parts of the body containing electronic implants/prostheses, on areas of malignancy, or over the lower back or abdomen during pregnancy.
- LFU has demonstrated bacterial killing during wound débridement via cavitation. This effect is unique to UGD

devices and should be considered when managing contaminated and critically colonized wounds.

- Contact devices are associated with thermal tissue effects, and clinicians should be skilled in UGD technique.
- As with all sharp débridement procedures, consideration and management of pain are paramount.

Biological

The group of biological modalities also includes a large number and variety of technologies and is a category that has experienced significant change, growth, research, and development. This progress includes both the actual technologies as well as our way of handling them from a nomenclature, organizational, and reimbursement perspectives. The activities of the Alliance of Wound Care Stakeholders provide a good illustration.[125,126] The alliance is a nonprofit multidisciplinary trade association constituted of professional societies with interest and clinical involvement in wound care (i.e., the American Professional Wound Care Association, the American College of Hyperbaric Medicine, the Association for the Advancement of Wound Care, and the Wound, Ostomy and Continence Nurses Society) and patient organizations whose mission is to promote quality care and access to products and services for people with wounds through effective advocacy and educational outreach in the regulatory, legislative, and public arenas. This group closely follows fiscal and regulatory policy, intervening with clinically appropriate commentary and recommendations. Historically, most of the advanced biological therapies were collectively referred to as tissue-engineered skin substitutes. Tissue engineering is the development of materials that combine novel substances with living cells to yield functional tissue equivalents/skin substitutes.[19] The term skin substitutes has become obsolete not only due to inaccuracy of the descriptor but also as it has caused confusion in discrimination between devices as well as difficulty for coding, billing, and reimbursement. The alliance via consensus recently proposed a new term for this category, cellular- and tissue-based products (CTPs). This new lexicon has been adopted by many professional societies as well as regulatory agencies.

For educational purposes, this discussion of biological therapies subdivides CTPs in to bioactive materials, biosynthetic matrices and grafts, biopharmaceuticals, and biotechnologies.[32]

Bioactive Materials

Bioactive materials are those derived directly from human tissue sources. Some of the technologies included in the group include Cultured Living Allografts (sometimes still referred to as bioengineered skin substitutes), Human Amnion/Chorion Membrane Allografts (HACMs), Autologous Platelet Grafts, epidermal grafts, and Autologous Dermal Matrix.

Cultured Living Allografts

Technology has spawned a new generation of biological materials to advance wound healing and provide all the characteristics of natural skin. Skin substitutes were originally designed to replace autografts, which are the harvesting of a patient's own skin to apply to a burn or wound.[9] Current devices may be composed of epidermal cells, dermal cells, or both supported by a biodegradable matrix.[9] Products containing both epidermal and dermal cells are referred to as bilayered.[9]

Two products containing living cells are approved for use in the United States, Apligraf and Dermagraft. Both contain living cells derived from neonatal foreskin. A single foreskin can produce over 200,000 units of the product.[26] Apligraf is bilayered consisting of a scaffold of type I bovine collagen that contains living human dermal fibroblasts and an overlying cornified epidermal layer of living human keratinocytes. Apligraf doesn't contain Langerhans' cells, melanocytes, or endothelial cells, perhaps explaining why it isn't clinically rejected. Dermagraft is composed of cryopreserved human fibroblasts on a polyglycan scaffold indicated for use in the treatment of full-thickness diabetic foot ulcers.[127,128] When Dermagraft is placed on an ulcer, the mesh material is gradually absorbed, and the human cells grow into place, replacing the damaged skin.[128] Both products are tested extensively for infectious agents. Cultured Living Allografts are not recommended for use on infected wounds or wounds with sinus tracts and should not be used in patients who are sensitive to bovine products.[128]

Cultured Living Allografts are applied by a clinician, typically in the wound care clinic, and several applications may be needed before healing is attained. Prior to application, excellent wound bed preparation is mandatory. Wounds are débrided, moisture balance maintained, and infection monitored. The wound may be cultured prior to the procedure. Appropriate oral antibiotic therapy or antimicrobial wound dressings maybe used to prevent "losing" the graft in the setting of heavy or critical colonization. The graft site must be

protected from injury; the secondary dressing is changed without disturbing the graft site.

Cultured Living Allografts have demonstrated improved outcomes over standard of care (SOC) in several randomized controlled trials in patients with diabetic foot ulcers and venous insufficiency.[129,130] This level of evidence has not been matched by many of the other products discussed in this section, and this has translated to widespread clinical use of these allografts.

Human Amnion/Chorion Membrane Allografts

HACMs have been used in surgical reconstruction for over 50 years yet have only recently entered the wound care clinic. HACMs are harvested from human placenta or umbilical cord and processed for use in wounds with the intent to promote healing. HACMs have been widely used for burns where extensive areas of skin have been damaged. These products contain either both amnion and chorion tissue or single-layer amnion alone. HACM tissues are nonimmunogenic, reduce inflammation and scar formation, contain relatively high levels of growth factors, and have been shown to stimulate cellular proliferation as well as promotion of mesenchymal stem cell migration.[131,132] Tissues are harvested during planned caesarian section deliveries and undergo extensive maternal and tissue microbiological and viral screening including HIV-1 and HIV-2 antibody, hepatitis B core antibody, HIV type 1 nucleic acid test (NAT), hepatitis B surface antigen, HTLV-1 and HTLV-2 antibody, hepatitis C antibody, serological test for syphilis, CMV total antibody test, and hepatitis C virus NAT. Several products are marketed and differ based on layers of amnion–chorion tissue used and variations in proprietary tissue processing.

The literature reports excellence healing outcomes can be gained with the use of HACMs. One study of DFUs compared HACM with SOC versus SOC alone. The authors reported 92% healed in 6 weeks compared to 8% for control, with an average of 2.5 graft implants used to closure.[133] Early reports and trials suggest that this technology may have a significant positive impact on the management of chronic wounds.

Epidermal Grafts

Epidermal grafting is the application of negative pressure to the skin to create little fluid-filled blisters (suction blistering), during which the lamina lucida of the skin is cleaved from the underlying layers. This separates the epidermis from the dermis. The blisters are then cut and emptied, and the loose skin is transferred side by side to the nonhealing wound. Epidermal grafting was first reported in 1964 to treat vitiligo as the epidermal grafting transfers melanocytes as well as keratinocytes.[134] The procedure is painless and allows for the efficient transfer of multiple epidermal micrografts to the recipient wound bed. The epidermal grafts are uniform in size and contain basement membrane components and keratinocyte-associated growth factors. Epidermal grafting has been suggested to be an excellent option for patients with clean granular wounds to hasten epithelialization and eliminate the need for operative intervention and split-thickness skin grafting. Clinicians skilled in epidermal grafting report painless, rapid healing of donor site especially when compared to standard skin grafting procedures (Fig. 9-22).

Figure 9-22. Xenograft matrix. (Photos courtesy of J.A. Niezgoda.)

Autologous Dermal Matrix

Autologous Dermal Matrices (ADMs) are allograft scaffolds, which are produced by harvesting human skin. The dermis is then minimally processed to remove the epidermis and all cellular components but maintain ECM structure using septic processing without terminal

sterilization. The finished product is lyophilized and packaged for storage and shipping. In this freeze-dried state, the product must be rehydrated prior to clinical application. This technology has been available for clinical use for several years from a variety of vendors (Fig. 9-23).

Figure 9-23. Platelet-rich plasma. (Photo courtesy of J.A. Niezgoda.)

Practice Essentials

- Meticulous wound bed preparation is paramount to good outcomes when using any biological material including Cultured Living Allografts, epidermal grafting, or ADMs.

- Patients with plantar surface wounds mandate aggressive off-loading after grafting.
- Watch for signs and symptoms of infection.
- During follow-up after implantation, it is not necessary to débride the yellow caramelized crust at the edges or in the wound; this is likely graft incorporation and must be distinguished from nonviable tissue.

Practice Point

To date, no biological material is able to completely replicate normal, uninjured skin.[9]

Biosynthetic Matrices/Xenografts

Biosynthetic matrices and xenograft materials constitute a group of technologies where the product is either produced synthetically or created using nonhuman animal tissues (typically bovine or porcine). These products are decellularized and sterilized, maintaining a rich collagen matrix, which is applied to the wound base to serve as a scaffold and orchestrate healing via dynamic reciprocity. The finished product is lyophilized and must be rehydrated. Collagen is typically cross-linked.

The xenograft matrix provides a three-dimensional, bioresorbable ECM (see Fig. 9-22) that is incorporated and absorbed into the wound. These matrix grafts are indicated for the management of partial- and full-thickness wounds, pressure ulcers, venous ulcers, chronic vascular ulcers, diabetic ulcers, trauma wounds (abrasions, lacerations, second-degree burns, skin tears), draining wounds, and surgical wounds (donor sites/grafts, post-Mohs' surgery, postlaser surgery, and wound dehiscence). Clinical data and randomized studies demonstrate improved healing over SOC in a variety of chronic wounds including diabetic foot ulcers, venous ulcers, and surgical wounds. A prospective, randomized, multicenter trial evaluated the effectiveness of SIS xenograft plus standard of care (SOC with compression therapy) versus SOC alone in 120 patients with chronic venous insufficiency and lower leg ulcers of greater than 1-month duration. At 12 weeks, the xenograft group demonstrated 55% healing compared to 34% of the SOC group ($P = 0.0196$).[135]

Biopharmaceuticals

Biopharmaceuticals are distinct from chemically synthesized pharmaceutical products. A pharmaceutical drug (medicine or medication and officially medicinal product) is any chemical substance formulated or compounded as single active ingredient or in combination with other pharmacologically active substance. Pharmaceuticals are typically engineered macromolecular products, and although they can include protein-based and nucleic acid–based drugs, which are sometimes called medicinal products or therapeutic biological products, they are chemically synthesized and not of biological origin. Synthetically derived compounds can therefore be distinguished from biopharmaceuticals, which are usually directly extracted from a biological source like blood or blood components.[136] Examples of biopharmaceuticals include vaccines, blood or blood components, allergenics, recombinant therapeutic proteins, and growth factors.

Growth Factors

Growth factors, also referred to as cytokines, are proteins (polypeptides) that occur naturally in the body. They are found primarily in platelets and macrophages. Currently, there is only one growth factor available in the United States, platelet-derived growth factor (PDGF). Several other types of growth factors are being studied in clinical trials, and many are already available for clinical use outside the United States. These include epidermal growth factors, transforming growth factors, fibroblast growth factors, and vascular endothelial growth factors (VEGFs). From a clinical and research perspective, growth factors can be categorized into two major groups: single growth factors manufactured through recombinant DNA technology (PDGF) and multiple growth factors secured from human platelet releasate.[24]

Regranex (becaplermin) Gel 0.01% is a recombinant PDGF that is available by prescription and is indicated for the treatment of lower extremity diabetic neuropathic ulcers that extend into the subcutaneous tissue or beyond and have an adequate blood supply. Regranex has been available for many years and has extensive clinical experience. It has been found to be efficacious in the management of diabetic ulcers and in granulating wounds but has recent concern raised secondary to a postmarketing retrospective cohort study, which reported an increased rate of mortality secondary to malignancy that was observed in patients treated with three or more tubes. The manufacturer has subsequently issued the recommendation that Regranex should only be used when the benefits can be expected to outweigh the risks and should be used with caution in patients with known malignancy.[137]

Practice Point

Becaplermin gel (Regranex) must be kept refrigerated and used with caution in patients with known malignancy.

Platelet-Rich Plasma

Platelet-rich plasma (PRP) is the exogenous application of autologous platelet-derived concentrate. PRP contains multiple growth factors. Unlike becaplermin, which is produced by the manufacturer and comes ready to use, PRP is prepared at the point of care for the patient. Blood taken from the patient is mixed with a small amount of anticoagulant citrate dextrose, and the solution is then centrifuged to separate the platelets and serum.[138,139] The concentrated platelets (see Fig. 9-23) are then placed into the specially provided syringe that mixes the concentrate as it is applied to the prepared wound bed as a gel.[138,139]

Biotechnologies

This final category of biological advanced therapies includes genetic transfer, stem cell therapies, and other progressive and novel technologies. Most of these therapies continue to be developed under research protocols with limited clinical utilization. While early data are intriguing and even compelling in some case, much more work is required to fully integrate these modalities in clinical algorithms with corresponding reimbursement.

Stem cell therapy is the process of delivering autologous stem or progenitor cells directly to wounded tissues to differentiate and replace deficient cells such as fibroblasts, endothelial cells, and myocytes or to produce proangiogenic proteins, which stimulate angiogenesis tissues for the purpose of enhancing wound healing. Stem cells are biological cells found in all multicellular organisms defined by two characteristics: (a) division, allowing mitosis or mitotic division and self-renewal to produce more stem cells, and (b) differentiation, creation and formation of diverse specialized cell types. While embryonic cells are called pluripotent with the ability to differentiate into multiple cell types, adult or somatic stem cells (the type used in wound care applications) are called progenitor cells and have limitations on their ability to differentiate and replicate. Clinically, stem cells are harvested from bone marrow, adipose tissue, or blood and processed via cell separation using high-speed centrifuge techniques. The isolated stem cells are then applied clinically using tissue injection or grafting. Literature data in wound care are early and sparse but studies have reported improved healing as well as synergy with HBOT.[140,141]

Pharmaceutical

Oxygen Therapy

As defined above, a pharmaceutical drug is any chemical substance formulated or compounded as single active ingredient or in combination with other pharmacologically active substance. Although oxygen is a critical element to living organisms as it drives nearly all biological processes, it does meet the criteria of a pharmaceutical as a chemical substance when administered for therapeutic purposes. Oxygen when utilized for the purpose of wound healing is provided as a prescriptive device, a pharmaceutical, and therefore is discussed separately from other biological therapies. There are various types of oxygen therapy available for the management of acute and chronic wound conditions. This section will discuss hyperbaric oxygen in detail based on the widespread use within the wound care community and then briefly discuss several types of topical or transdermal oxygen devices.

Hyperbaric Oxygen Therapy

Definition

HBOT is defined as treatment during which a patient inhales 100% oxygen while enclosed in a pressurized chamber exceeding 1.4 atmospheres absolute (ATA). HBOT has been used worldwide for more than 50 years in the treatment of many chronic nonhealing wounds and ulcers.[142] Treatments are administered either in a multiplace chamber (Fig. 9-24A) occupied by several patients or in smaller monoplace chamber (Fig. 9-24B), containing only one patient at any one time. In multiplace chambers, patients are treated simultaneously in an environment of pressured air, while breathing pure oxygen delivered via mask or hood tent. In a monoplace chamber, a single patient is placed in a pressurized environment of pure oxygen. For routine conditions, HBOT is typically administered between 2.0 and 2.5 ATA, with 90 minutes of oxygen breathing, interrupted briefly by an “air break” (5 to 10 minutes of air breathing). The safety record of HBOT in this country is excellent, and the technology has become a vital part of the growth of comprehensive wound care programs across the nation.[67,143]

A

Figure 9-24. Hyperbaric chambers. **(A)** Hyperbaric multiplace chamber. (**B)** Hyperbaric monoplace chamber. (Photo courtesy of J.A. Niezgoda.)

Mechanism of Action of HBOT

An uncompromised patient, breathing air at sea level, will typically have an arterial pO_2 of 100 mm Hg. The majority of this oxygen is carried bound to hemoglobin with only a small percent of oxygen physically dissolved in the plasma. The normal diffusion radius for an oxygen molecule is 64 microns at the end arteriole and about 36 microns from the venous system where the pO_2 might be as low as 34 mm Hg. However, this same patient breathing 100% oxygen at 3 ATA will have an enormous increase in the amount of oxygen physically

dissolved in the plasma (6.9 volumes percent) compared to the volume of oxygen dissolved at 1 ATA (0.3 volume percent) (Fig. 9-25). Plasma pO_2 values well in excess of 1,500 to 1,800 mm Hg are routinely achieved in hyperbaric patients. In this setting, the hemoglobin never desaturates, and thus, hemoglobin plays a minimal role in the transport of oxygen. Tissue oxygen partial pressures can reach supraphysiologic oxygen levels of 250 to 300 mm Hg, far above the normal 30 to 40 mm Hg when breathing 1 ATA air. This 18- to 20-fold increase in dissolved oxygen establishes a tremendous gradient between the plasma and the peripheral tissues and provides tissue hyperoxygenation and stimulation of neoangiogenesis and drives the other physiological benefits of HBOT.[67,144]

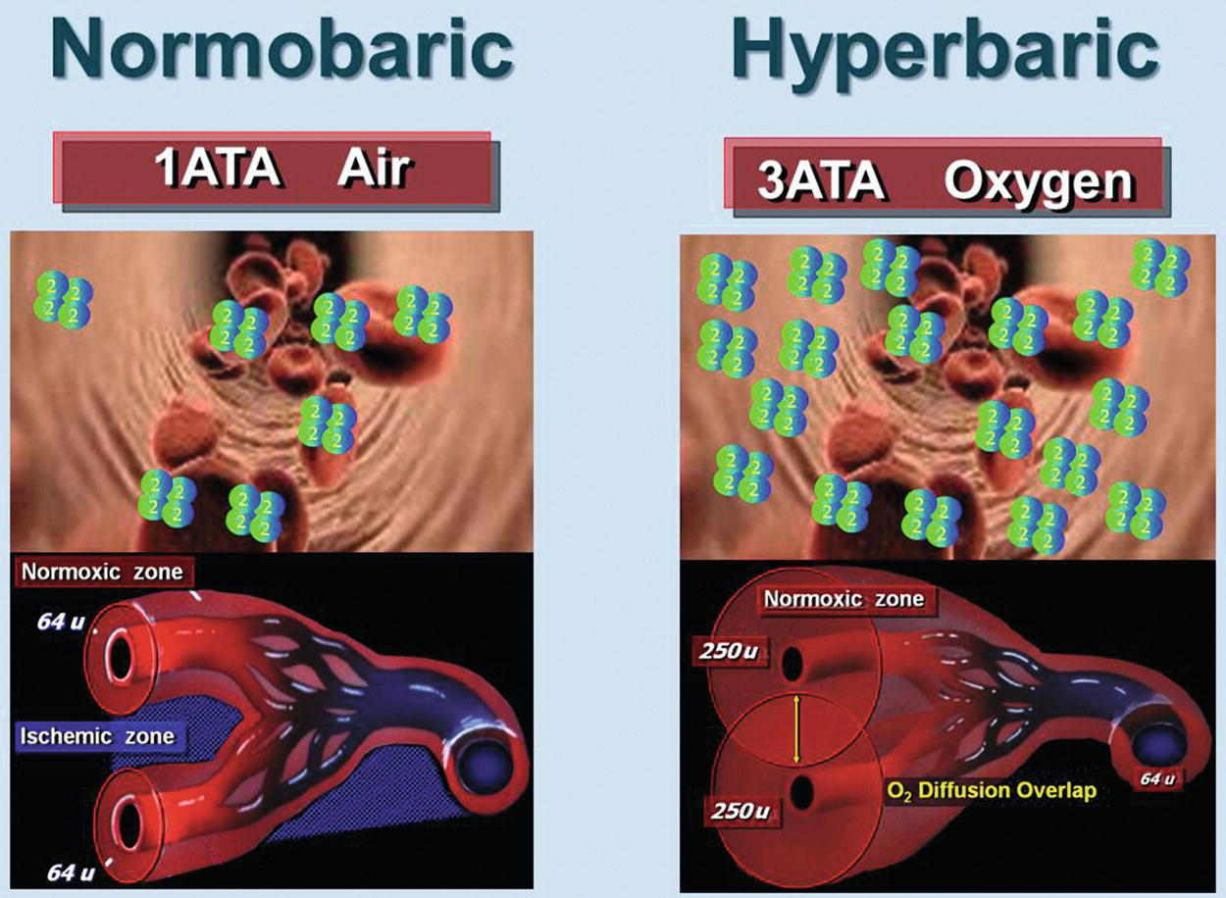

Figure 9-25. Hyperbaric physiology. (Photo courtesy of J.A. Niezgoda.)

Physiological Effects of HBOT

Hyperbaric oxygen has been demonstrated to deliver several physiological effects that can have a positive impact on wound healing and well as other non–wound-related indications, such as carbon monoxide poisoning and decompression illness.[143]

- Hyperoxygenation of ischemic tissue: Chronic wounds are frequently hypoxic. HBOT increases tissue partial pressure of oxygen (pO_2), correcting wound hypoxia intermittently.[105] It then allows for acceleration of the wound healing process through a series of actions that continue long after the HBOT session has ended and tissue oxygen levels have returned to pretreatment values.[145–147]
- Angiogenesis: HBOT can induce angiogenesis and neovascularization in hypoxic tissues via induction and increased expression of VEGF. Studies demonstrate that VEGF levels in hypoxic wounds rose by approximately 40% within 5 days of starting HBOT but then dropped to control levels within 3 days of stopping the treatment. In addition to an increase in angiogenesis, there was a 20% increase in wound bed perfusion following 10 days of HBOT as demonstrated via laser Doppler imaging.[148,149]
- Vasoconstrictive effects of oxygen: Vasoconstriction takes place in both arterial and venous vessels, which reduces edema and congestion while the amount of oxygen supplied by the plasma is increased.[150]
- Improved wound metabolism: The elevation in pO_2 promotes wound healing by directly enhancing fibroblast replication, collagen synthesis, and the processes of neovascularization and epithelialization.[145,151]
- Up-regulation of growth factors: HBOT causes the up-regulation of cytokines, including PDGF and VEGF, thereby stimulating cellular repair and regeneration as well as enhancing angiogenesis.[152,153]
- Antibacterial effects: The increase in available oxygen enhances the leukocyte bactericidal effect, including the killing of aerobic gram-positive (*S. aureus*) and gram-

negative organisms in addition to being cytotoxic to anaerobes. Neutrophils or polymorphonuclear cells (PMNs) require oxygen for phagocytosis and killing of bacteria via oxygen-free radical formation and other oxidative mechanisms. Should the oxygen tension fall below 30 mm Hg, the efficiency of bactericidal action of PMNs decreases dramatically, leaving the patient at higher risk for infection.[153]

- Antioxidant effect: HBOT has been demonstrated to have a positive impact on limiting the deleterious effects of ischemia–reperfusion injury. It blocks guanylate cyclase, the trigger molecule for beta-2 integrin that forms the leukocyte adhesion molecule. HBOT also blocks the action of the intracellular ICAM adhesion molecules that cause any leukocytes that become adherent to the capillary wall to bore through into the tissues surrounding the vessel, releasing the superoxide anion, proteinases, and elastase that liquefy tissue following crush injury and other forms of ischemia. Thus, following ischemia or sepsis, when the patient is treated early with HBOT leukocytes, do not adhere to the capillary walls to block circulation or cause lipid peroxidation.[154,155]

Clinical Indications for HBOT

The clinical indications for HBOT are provided based on review of current available evidence by expert consensus of professional societies. The American College of Hyperbaric Medicine and the Undersea and Hyperbaric Medical Society list the following indications[156,157]:

- Air or gas embolism
- Carbon monoxide poisoning
- Carbon monoxide poisoning complicated by cyanide poisoning
- Clostridial myositis and myonecrosis (gas gangrene)
- Crush injury, compartment syndrome, and other acute traumatic ischemias
- Decompression sickness

- Arterial insufficiencies (enhancement of healing in selected problem wounds)
- Central retinal artery occlusion
- Severe anemia
- Intracranial abscess
- Necrotizing soft tissue infection
- Osteomyelitis (refractory)
- Delayed radiation injury (soft tissue and bony necrosis)
- Compromised grafts and flaps
- Acute thermal burn injury
- Idiopathic sudden sensorineural hearing loss

Despite the opinion of these professional societies, many payors including CMS have different approved indications for reimbursement purposes. The hyperbaric clinician must be aware of these discrepancies in interpretation of the literature and evidence.

Special Considerations

The physiological effects of systemic oxygen on the human body are more similar to a drug than a physical modality, and, like any drug, there is the risk of overdose (oxygen toxicity) and side effects (primarily barotrauma).[7,145,158,159] To ensure appropriate utilization and patient safety and to minimize the potential of adverse outcomes, HBOT must be supervised by a hyperbaric-certified physician with expertise and training in this highly specialized treatment modality. The American College of Hyperbaric Medicine has published credentialing guidelines and training recommendations and endorses the hyperbaric certification examinations offered by the American Board of Wound Healing (ABWH).[160]

Summary

HBOT is an advanced wound care technology that can have a significant positive and powerful effect in the management of patients with compromised wound healing, especially when the primary deficit is tissue hypoxia. When patients are carefully selected and appropriately managed with HBOT as part of a comprehensive wound

care strategy, wound healing enhancement can be realized and limb loss avoided.

Practice Essentials

- HBOT is an adjunctive therapy accompanying good wound care practices.
- Patients should undergo aggressive arterial evaluation and revascularization when possible prior to the initiation of HBOT.
- Clinicians must be trained and educated in the physiology, mechanisms, and indications for HBOT, understand the importance of patient selection and safety, and seek certification to ensure appropriate utilization and optimal outcomes.
- Treatment cost and insurance approval should be considered before initiating HBOT.

Practice Point

Carefully monitor the blood glucose of patients with diabetes before HBOT. Blood glucose levels have been known to drop precipitously when patients are in the HBOT chamber, putting them at risk for seizures or other complications from hyperglycemia.

Transdermal Oxygen/Topical Oxygen Therapy

Transdermal or topical oxygen therapy is the application of oxygen to the surface of a wound. Historically, this has been accomplished by placing the affected limb in an enclosed acrylic or plastic box and filling it with oxygen. Topical oxygen is frequently (and inappropriately) called “topical hyperbaric oxygen.” Use of this term simply adds confusion and is a misapplication of the word hyperbaric. Hyperbaric oxygen is defined with the inhalation of 100% oxygen while the entire patient is enclosed within a chamber at pressures of at least 1.4 ATA or greater neither of which is achieved by extremity devices. The stratum corneum in intact skin represents a significant barrier to oxygen diffusion from the atmosphere. However, in a patient with a wound, the absence of the dermis more easily allows for direct oxygenation via diffusion. Multiple studies report that topical oxygen that dissolves in tissue fluids is bacteriostatic and stimulates angiogenesis and wound healing.[161] A novel device that provides direct transdermal tissue oxygenation has been produced and studied. OxyBand Wound Dressing (OxyBand Technologies, St Louis, MO) is a transdermal therapy that delivers oxygen to a wound for up to 5 days following application. The oxygen is contained in a reservoir and diffuses into the wound base continuously at a rate controlled by a propriety semipermeable membrane (Fig. 9-26). A recent prospective, randomized, controlled study of burn patients undergoing harvesting of two donor sites reported significantly faster wound healing for OxyBand (9.3 ± 1.7 days; control, 12.4 ± 2.7 days [$P < 0.001$]) as well as lower pain scores in the OxyBand group ($P < 0.01$).[162]

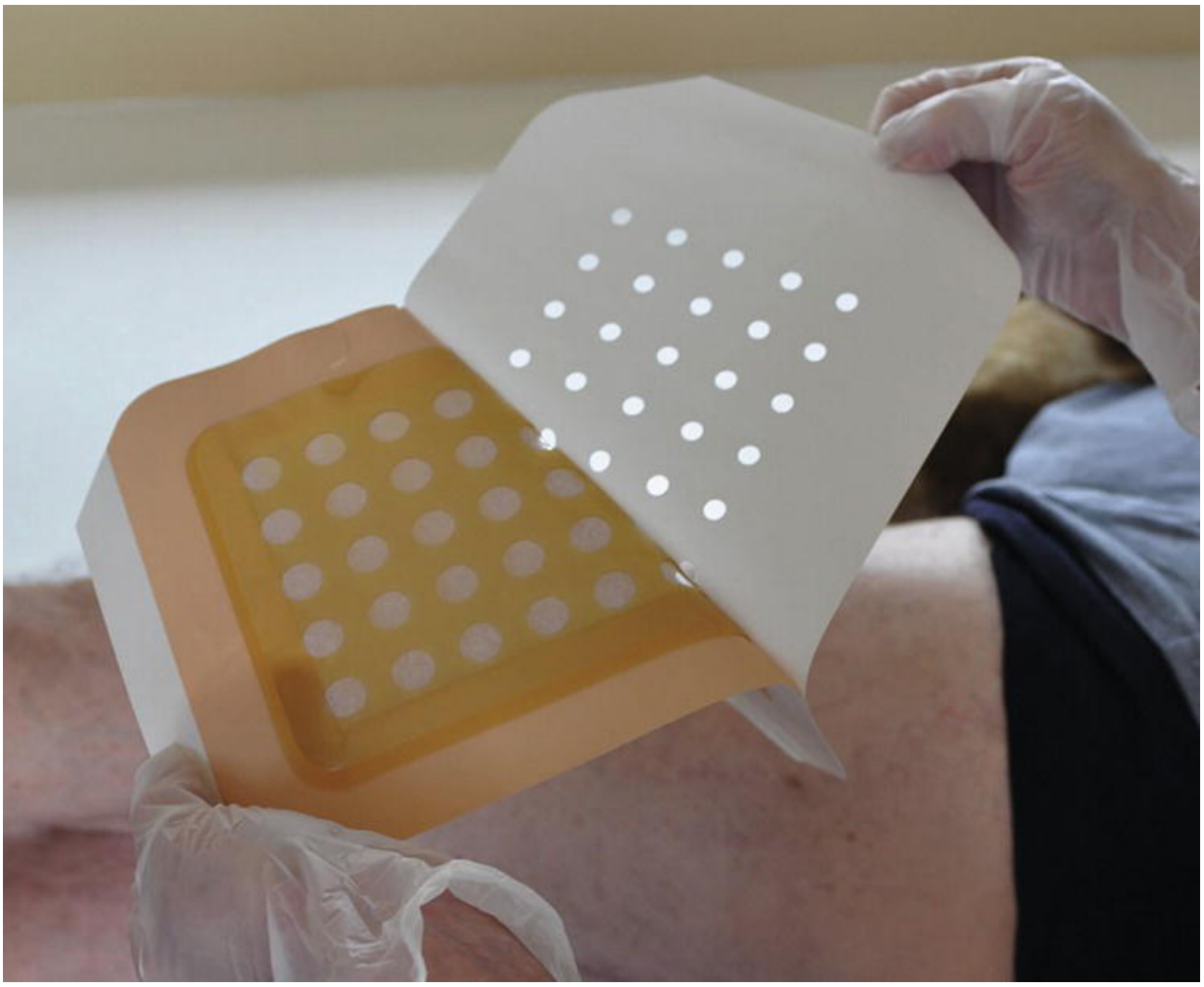

Figure 9-26. Transdermal oxygen therapy. (OxyBand Wound Dressing TM, OxyBand Technologies, St Louis, MO.)

Oxygen therapy plays a significant role in wound healing. The wide use of hyperbaric oxygen has been recently challenged on the basis of overutilization and cost as well as on clinical efficacy. In our ever-changing world of wounds, these issues must and will be addressed. Alternative oxygen delivery systems should be considered and warrant additional clinical investigation.

- Frequency of dressing change varies among antimicrobials.
- Antimicrobial dressings are used in wounds with high bacterial bioburden or in wounds that are critically colonized

to prevent wound infection.

- Antimicrobial dressings may be used under compression wraps to prevent infection in wounds with a high bacterial bioburden.

Scar Management

Patients, clinicians, and researchers are all concerned about scar appearance. Progress has been made in our understanding of the mechanisms involved in producing an exaggerated scar. The scientific principles for scar management and minimization—support, controlled inflammation, adequate hydration, and remodeling/maturation of collagen[163,164]—form the basis of product selection for scar control strategies (Table 9-7).

Table 9-7 SCAR Acronym for the Practical Application of Principles for Scar Management

Principle	Pathophysiology	Proven Agents (Examples)
Support	• Vector forces increase the production of collagen. • All scars, especially long scars, need support in areas where vector forces continually pull on the scar.[163–168] For example, in a presternal chest area wound, neck, shoulder, and arm movements as well as weight from the breasts generate vector forces on the scar. (See Fig. 9-29B.)	• Microporous tape is the best form of scar support.[165,169,170] • To ensure that there is consistent support for the scar, the tape must be applied longitudinally along the scar path and not at right angles. (See Fig. 9-29A.) • Leave the tape in place for several days until it spontaneously separates from the skin. • Premature removal of the tape results in skin stripping, which sets up inflammation with negative consequences on the scar. • Small scars in some areas (face) may not need support.
Controlled inflammation	Because excessive inflammation results in exaggerated scars, controlled inflammation is a sought-after principle in scar management.	• Topical application of olive oil compounds (phenol compounds such as oleuropein in newly pressed olive oils) has anti-inflammatory activity and antimicrobial effects.[171,172] • Oleuropein stimulates proteasome function and fibroblast formation of new collagen.[173] Proteasomes prevent collagen from clumping as they up fragmented protein particles, including fragmented collagen, a process critical to the prevention of clumped collagen. Thus, stimulation of proteasomes and inhibition of inflammation are extremely advantageous to the process of scar maturation.
Adequate hydration	• Hydration of the scar surface is the basis of action of 90% of scar management systems on the market. • Most oils (tissue oils), lotions, and creams have beneficial effects on scars purely on the basis of their hydrative capacities.[174–176] Although this is obviously beneficial, it is limited in terms of the outcome it can produce and affects only one area of scar control. • Normal skin has a mature stratum corneum characterized by minimal transepidermal water loss (TEWL). • Dehydration of the stratum corneum initiates signaling to keratinocytes. These keratinocytes are stimulated to produce cytokines, which activate dermal fibroblasts to synthesize and release collagen. Excessive collagen production leads to abnormal scarring.[177]	• The most effective barrier to TEWL and stratum corneum breach is silicone, in the form of either sheeting or gels (dimethicone).[177–179] • In addition, gel derived from the plant *Bulbine frutescens* has been found to be effective as a hydrating agent; the glycoproteins of this plant extract are large and remain on the surface of the skin long enough to produce effective hydration of the skin.[165,166]

Principle	Pathophysiology	Proven Agents (Examples)
Remodeling/ maturation of collagen	• The quicker the scar matures, the less chance there is of hypertrophy. • Collagen maturation goes through phases, with type III collagen being present in greater levels in the early scarring phase. • As the scar matures, the ratio of type III to type I collagen returns to normal levels.[180] Thus, any agent that encourages a return to stable ratios is advantageous to scar outcome. • Transforming growth factor (TGF)-β is the prototype of a protein superfamily that has been recognized as the major fibroproliferative and collagen-stimulating agent involved in excess scarring (particularly TGF-β_1). Many isoforms of the protein exist, most of which share the same fibroproliferative properties. One isoform (TGF-β_3), however, appears to have a protective effect against excess collagen formation counteracting the TGF-β_1 effects.[181]	• Extracts of the *Centella asiatica* plant increase levels of mature collagen and encourage normalization of collagen ratios.[180–183] • Purified extracts (triterpenic fractions, including asiaticoside) isolated from *C. asiatica* have been shown to induce type I collagen synthesis in human dermal fibroblast cells[180–182] • Asiaticoside down-regulates TGF-β_1 expression and up-regulates TGF-β_3 expression and is also capable of decomposing the products of type I collagen, contributing to the reduction of hypertrophic scar formation.[181] • Laboratory evidence of asiaticoside efficacy was demonstrated in the rabbit ear model, one of the only consistent animal models producing hypertrophic scarring.[183,184]

Adapted with permission from Widegerow, A.D. “Scar Management: The Principles and Their Practical Application.” *World Council of Enterostomal Therapists Journal* 31(1):18-21, 2011.

Scar control does not rely on a single modality but rather on a number of proven factors whose combination results in a good outcome. Controlling scar formation is an important part of wound management practice. Widgerow and colleagues[163–165] have described a patented process of applying a cream/gel that contains antiscar active agents (*Centella asiatica*, oleuropein, dimethicone, *Bulbine frutescens*) to the surface of microporous tape. This process has been used successfully for scar management. Within 2 minutes, the active agents in the gel are absorbed through the tape and onto the scar tissue. The saturated tape continues to work as an occlusive scar dressing. The tape remains in place during patient bathing and is only replaced when it separates from the skin (usually in 3 to 5 days). Gel is reapplied to the tape surface twice a day until scar maturation (white color) begins to occur. Once the scar is maturing well (usually 6 weeks), use of the tape can be stopped and the gel applied directly to the scar.[165] In a comprehensive trial,[164] 170 scars were assessed based on the SCAR acronym (Fig. 9-27). Hypertrophy was prevented in more than 80% of

cases. Hypertrophy and scar exaggeration are seen in 60% to 80% of cases when there is no scar management.[166]

A

B

Figure 9-27. (A) Tape applied longitudinally along the direction of the scar. White or flesh-tone tape may be used, with the same effect. **(B)** Untreated hypertrophic scar in the presternal area; multiple vector forces are working against this scar. **(C)** The mature treated

abdominal scar is flat, white, and nonreactive (note that it is normal for most scars to become hypopigmented to some extent). Compare this scar with the untreated scar. **(D)** Keloid scarring, which grows like a tumor and flows over the scar boundary, occurs wherever the skin is breached. Its pathogenesis and treatment are different from those of hypertrophic scars.

Although often confused as the same, keloid scars are different from hypertrophic scars. Keloid scarring usually has a genetic component, where type 1 collagen is produced in a tumorlike fashion with uncontrolled growth of scar tissue. The typical wound history includes a well-managed noninfected wound that progressively increases in size and extends over the wound boundary. These scars may be painful, sensitive, and extremely uncomfortable. Treatment (often radiotherapy) is unpredictable and unsatisfactory. The SCAR principles are not applicable to keloid scarring. The different mechanisms between hypertrophic and keloid scars require different product approaches.

Summary

The selection of appropriate wound treatment options must be patient centered and driven by the specific goals of care. A succinct overview of the important characteristics and use of wound dressing products is provided (Table 9-8).

Table 9-8 Wound Dressing Categories

Key—Exudate amount: ◊ Dry ♦ Light ♦♦ Moderate ♦♦♦ Heavy

Generic Category	Description/ Composition	Trade Names		Indications	Advantages and Benefits	Disadvantages
Transparent film ◊	Polyurethane or polymer membrane with porous adhesive layer that varies in thickness and allows oxygen to pass through and moisture vapor to escape	3M Tegaderm Bioclusive DermaView Gentell MVP Hydrofilm OPSITE Suresite Transeal		Donor sites Primary and secondary dressings Partial-thickness wounds Pressure injuries, stages 1 and 2 Superficial burns Peripheral IVs Abrasions	Wound inspection Impermeable to external fluids and bacteria Conformable Promote autolytic débridement Prevention/reduces friction Change every 5–7 days or prn if leakage noted Numerous sizes available Waterproof	Nonabsorptive May adhere to fragile skin Not for draining wounds Fluid retention may lead to maceration of periwound area. Third-degree burns
Hydrocolloid ♦ ♦♦	Occlusive or semiocclusive dressing that consists of gelatin, pectin, and carboxymethylcellulose Impermeable to bacteria and other contaminants Wafers are self-adhering and are available with or without an adhesive border Available in a wide variety of shapes and sizes	3M Tegasorb Comfeel Plus CovaWound DermaFilm DuoDERM CGF Exuderm FlexiCol Gentell	Nu-Derm PrimaCol ProCol REPLICARE Restore	Pressure Injuries, stages 2 and 3 Partial- and full-thickness wounds Preventive dressing for high-risk friction areas Secondary dressing or under taping procedures First- and second-degree burns	Facilitates autolytic débridement Self-adherent Impermeable to fluids/ bacteria Conformable Reduces wound pain Thermal insulation Long wear time—3–7 days, depending on exudate	Contraindicated with muscle, bone, or tendon Not recommended for heavily draining wounds, sinus tracts, or fragile skin May be difficult to remove Contraindicated for third-degree burns

Hydrogel ◊ ◆	Water or glycerin based, nonadherent; contains 80% to 99% water; numerous sizes and forms (gels, sheets, strips, and gauze available)	3M Tegaderm Hydrogel Amerigel AquaSite Intrasite Gel PluroGel Prontosan Purilon Gel Regenecare Restore Gel	DermaGauze DermaSyn Elasto-Gel Gentell Hydrogel Skintegrity SoloSite TenderWet Woun'Dres Hydrogel	Pressure injuries, stages 2–4 Partial- and full-thickness wounds Abrasions Painful wounds Radiation tissue damage First- and second-degree burns Skin tears Necrotic wounds	Nonadherent Trauma-free removal/soothing to patient Rehydrates the wound bed Reduces wound pain Can be used with topical medications Can be used in cavities or tunnels Softens and loosens necrosis and slough 24–72 hours dressing change, depending on the form of gel	Some require secondary dressing to secure May macerate periwound skin Not recommended for heavily draining wounds Contraindicated for third-degree burns
Hydrogel with silver ◊ ◆	Controlled-release ionic silver	Anasept Gel DermaSyn Ag Gentell Hydrogel Ag Resta Silver Gel SilvaKollagen Gel SilvaSorb Gel Silver-Sept Gel		Infected wounds Highly colonized wounds	Antimicrobial Can be left on up to 3 days Nonadherent Rehydrates the wound bed	Sensitivity to silver Not recommended for use in conjunction with topical medications Will inactivate enzymatic débriding agents Same disadvantages as hydrogel dressings
Foam ◆ ◆ ◆ ◆ ◆	Hydrophilic polyurethane or gel film–coated foam, nonadherent layer absorptive wound dressing	Allevyn Aquacel Foam Biatain ComfortFoam CovaWound Cutimed DermaFoam HydraFoam HydroCell	Lyofoam Mepilex MPM Foam Optifoam PermaFoam PolyMem Proximel SiliGentle Tielle	Partial- and full-thickness wounds Pressure Injuries, stages 2–4 Surgical wounds Under compression wraps/stocking	Trauma-free removal Conformable, easy to apply and remove Frequency of dressing change depends on amount of drainage 3–5 days dressing change Available with adhesive and nonadhesive border in various shapes and forms	Not recommended for nondraining wounds Not recommended for dry eschar May require secondary dressing May macerate periwound area if not changed appropriately Contraindicated for third-degree burns
Foam with silver ◆ ◆ ◆ ◆ ◆	Controlled-release ionic silver	Acticoat Moisture Control Allevyn Ag Aquacel Ag Foam Biatain Ag ComfortFoam Ag CuraFoam Ag HydraFoam Ag Mepilex Ag PolyMem Silver Optifoam AG		Infected wounds Highly colonized wounds	Antimicrobial Some can be left on up to days Same advantages as foam dressings	Sensitivity to silver Must have exudate for silver to be released Will inactivate enzymatic débriding agents Same disadvantages as foam
Wound Absorptive Dressing						
Calcium alginate ◆ ◆ ◆ ◆ ◆	Nonwoven composite of fibers from calcium–sodium alginate, a cellulose-like polysaccharide, manufactured from brown seaweed; forms a soft gel when in contact with wound exudate	3M Tegaderm Alginate Aquacel (Hydrofiber) Algicell AlgiSite Biatain CalciCare DermaGinate Gentell Calcium Alginate Kalginate Kaltostat	Maxorb Extra (CMC Alginate) Melgisorb Nu-Derm Alginate Opticell Restore Simpurity Sorbalgon Sorbsan	Partial- and full-thickness wounds Pressure injuries, stages 3 and 4 Dehisced wounds Post-op wounds for hemostasis Sinus tracts, tunnels, or cavities	Trauma-free removal Can be used with tunneling and undermining Hemostatic properties for minor bleeding Change every day to every other day Available in sheets, ropes, and within other composite-type dressings	Contraindicated for dry eschar, third-degree burns, and heavy bleeding May require secondary dressing Gel may have odor during dressing change
Calcium alginate with silver ◆ ◆ ◆ ◆ ◆	Controlled-release ionic silver	Algidex Ag Algicell Ag CalciCare Ag DermaGinate Ag Gentell Calcium Alginate Ag KerraCel Maxorb Ab (CMC Alginate) Opticell Ag Restore Alginate with Silver Silverlon Calcium Alginate Sorbalgon Ag		Infected wounds Highly colonized wounds	Antimicrobial Change every 2 to 3 days or as needed Same advantages and benefits as calcium alginate dressings	Sensitivity to silver Must have exudate for silver to be released Will inactivate enzymatic débriding agents Same disadvantages as calcium alginate dressings

Composites ◆ ◆◆ ◆◆◆	Combination of two or more physically distinct products manufactured as a single dressing that provides multiple functions; such as a bacterial barrier, absorptive layer, and adhesion Usually, they are composed of multiple layers with a semi- or nonadherent pad that covers the wound May also include an adhesive border of nonwoven fabric tape or transparent film	3M Medipore 3M Tegaderm Absorbent Pad Alldress	Covaderm Plus Coverlet Covrsite DermaDress Dudress Film Top DynaGuard Leukomed Mediplus Mepore OPSITE Composite Repel Stratasorb Suresite Telfa Island Viasorb	Primary and secondary dressings for partial- and full-thickness wounds Pressure injuries, stages 2–4 Surgical incisions	Conformable Multiple sizes and shapes available Easy to apply and remove Most include adhesive border May be used with topical medications Frequency of dressing change dependent on wound type (check package insert)	Adhesive borders may limit use on fragile skin Some contraindicated for stage 4 pressure injuries (Check package insert) May not provide moist wound environment
Enzymatic débriders ◊ ◆◆ ◆◆◆	Prescriptive collagenase ointment that digests collagen	Collagenase/Santyl		To débride necrotic wounds, pressure injuries, post-op wounds	Collagen in healthy tissue is not attacked Nonsurgical method of débridement Requires daily dressing changes	Adversely affected by certain detergents, acidic solutions, and heavy metal ions such as mercury and silver
Collagen ◆ ◆◆ ◆◆◆	Major protein of the body. Dressing stimulates cellular migration and contributes to new tissue development derived from bovine, equine, porcine, or avian sources Some interact with wound exudate to form a gel	Biopad Biostep CellerateRx Gel/Powder ColActive DermaCol Endoform Fibracol Promogran Matrix Puracol Stimulen		Chronic nonhealing wounds Partial- and full-thickness wounds Pressure injuries, stage 3 and some stage 4 (check package insert) Surgical wounds	Absorbent, nonadherent Biodegradable gel Conforms well May be used in combination with topical agents 1–3-day dressing change	Contraindicated for third-degree burns and sensitivities to collagen or bovine products Not recommended for necrotic wounds May require rehydration
Collagen with silver ◆ ◆◆ ◆◆◆	Releases silver ions that are antimicrobial while collagen binds with MMPs (matrix metalloproteases) in chronic wound exudate	Biostep Ag ColActive Plus Ag DermaCol Ag Collagen Promogran Prisma SilvaKollagen Gel		Infected wounds Highly colonized wounds	Antimicrobial Change every 3–5 days as needed. Same advantages as collagen dressings	Sensitive to collagen and silver Will inactivate enzymatic débriding agents Same disadvantages as collagen dressings
Antimicrobial Therapies						
Nonadherent antimicrobial dressings that protect against bacteria and/or decrease bacterial load and bioburden				Helps to reduce infection and provide protection from common infectious bacteria and other contaminants	Decreases bacterial load in wound Can be used for the management of wound infections in acute or chronic wounds	Contraindicated in patients sensitive to ingredients described
◆ ◆◆	Cadexomer iodine impregnated: immediate and controlled release of cadexomer iodine	Iodoflex Pad Iodosorb Gel		Infected wounds—any type (pressure injuries venous, arterial, diabetic, or surgical wounds)		
◆ ◆◆ ◆ ◆◆	Controlled-release silver powder Polyhexamethylene biguanide impregnated (PHMD)	Arglaes Powder (Space between) Kerlix AMD		Partial- or full-thickness wounds Colonized, chronic nonhealing wounds	Provides protections against gram- and gram+ (including MRSA and VRE) Can be used in tunneling and undermining	
◆◆ ◆◆◆	Methylene blue and gentian violet Bacteriostatic foam dressing	Hydrofera Blue		Provides broad-spectrum antibacterial protection and helps protect against microorganisms		

Contact layer with silver ◆ ◆ ◆ ◆ ◆ ◆	Immediate and sustained release of ionic silver in a nonadherent primary dressing	3M Tegaderm Ag Mesh Acticoat Restore TRIACT Silver SilverDerm 7 Silverlon		Infected wounds Partial- or full-thickness wounds Colonized, chronic nonhealing wounds Under compression wraps/stockings Over grafts or skin substitutes	Inhibits growth of pathogens, especially antibiotic-resistant strains Antimicrobial action effective up to 3–7 days	Sensitivity to silver Secondary dressing required Must be removed and wound cleansed prior to MRI Not recommended for use in conjunction with topical medications Some may stain or discolor surrounding tissue due to silver turning black when it oxidizes Contraindicated with enzymatic débriding agents
Contact layer ◊ ◆ ◆ ◆ ◆ ◆ ◆	Nonadherent primary dressing that allows exudate to pass through Helps to reduce potential trauma to the wound bed	3M Tegaderm nonadherent contact layer Adaptic ColActive Transfer Comfitel Conformant 2 nonadherent contact layer Covrsite DermNet Drynet Wound Veil Mepitel N-Interface Physiotulle Telfa Clear Versatel		Partial- and full-thickness wounds, donor sites, skin grafts	May be used with topical medications Less trauma to the wound bed Prevents the outer dressing from adhering to the wound. Use in combination with other dressings to maintain moisture.	Secondary dressing is required.
Negative pressure wound therapy ◆ ◆ ◆ ◆ ◆	Noninvasive active therapy using localized negative pressure to promote healing	ActiV.A.C Engenex extriCARE Invia Kalypto PICO Prevena PRO-II Renasys SVED SNaP V.A.C		Moderate to heavy exuding wounds Partial- and full-thickness wounds Venous, arterial, diabetic ulcers, and dehisced wounds Pressure injuries, stages 3 and 4 Surgical wounds Flaps and grafts Acute traumatic wounds	Decreases edema Decreases bacterial colonization Increases blood supply and granular tissue formation Generally, dressing changed every 48–72 hours (specifics for dressing and equipment vary by manufacturer)	Healthcare worker needs training to apply and operate equipment May adhere to some wounds Not recommended for wounds with eschar Contraindicated for wounds with malignancy and untreated osteomyelitis
Advanced Wound Care Therapies						
Cellular- and/or tissue-based products ◆	– Nonviable cells, tissue-based human – Nonviable cells, tissue-based animal – Viable human cells, noncultured: intact tissue – Viable human cells, cultured in vitro: animal substrate – Viable human cells, cultured in vitro: synthetic substrate	Note—made it alphabetical. Can be divided into 2 sides Affinity AlloSkin Apligraf AmnioBand AmnioFill Biovance DermACELL Dermagraft EpiCord EpiFix GammaGraft Graftjacket Grafix MatriStem Mediskin Oasis Omnigraft PriMatrix Stravix Theraskin		Partial- and full-thickness wounds Venous and diabetic ulcers Granular wounds Burns Chronic wounds	Growth factors and/or collagen may be incorporated within the product Decreases wound healing time No donor site Decreases pain for many patients	Wound has to be granular Check manufacturer's directions for storage and shelf life Free of infection Review product description if patient has sensitivity to porcine- or bovine-derived products

Miscellaneous wound dressings	Medical-grade active *Leptospermum or Manuka* honey from New Zealand Multiple versions available to handle differing levels of exudate	Activon Dynarex L-Mesitran Gentell Honey Gauze Manuka Medihoney Therahoney		Infected wounds Highly colonized wounds Wounds with slough or necrotic tissue Pressure injuries, stages 2–4 Partial- and full-thickness wounds Venous ulcers Diabetic ulcers Burns—first- and second degree	Antibacterial barrier Autolytic débridement properties	Allergies to honey Mild stinging sensation may be present
	Sodium chlorite dressing consists of soft nonwoven material (viscose/polyester) impregnated with sodium chloride	Mesalt		Moderate to heavy exuding wounds of any type Infected wounds of any type	Facilitates cleansing of wounds with exudate, slough, or infection	Should not be used on dry wounds or minimally exuding wounds Should not be allowed to come in direct contact with exposed bone or tendon
	Balsam Peru, Castor Oil USP	Venelex		Pressure injuries, dehisced wounds Radiation injury	Balsam Peru is a capillary bed stimulant and a mild antiseptic that helps prevent bacteria from growing. Castor oil works as a lubricant to protect the tissue. Helps relieve pain Does not require a secondary dressing	Temporary stinging sensation BID application Prescription is required
Scar care	Self-adhesive silicone contained within a gel or flexible sheet Occludes the skin to hydrate the scar area	CICA-CARE MediClear Scar Mepiform		Hypertrophic scars, keloids, and closed wounds to prevent hypertrophic and keloid scars	Reusable, durable Conformable	Contraindicated in open or infected wounds Contraindicated over scabs and stitches May take 2–4 months to see some results
Becaplermin gel 0.01%	Genetically engineered human platelet-derived growth factors produced in yeast and then formulated into a gel	Regranex		Diabetic neuropathic ulcers	Stimulates fibroblast proliferation to increase granulation tissue	Should not be used on infected wounds or wounds with necrotic tissue Must be refrigerated

The products listed are representative of type and are not meant to be all-inclusive; indications, advantages, and disadvantages are some examples; more suggestions may apply.
Antimicrobials may be topical or within a dressing component. Source: Surgical, Mechanical, Autolytic or Enzymatic debridement methods © A. McIntosh, L. Montoya, January 24, 2020.

Selecting from the abundant number of wound dressings that are available today can pose a challenge. Tools such as wound product charts and enablers such as MEASURES© or NICE© give clinicians a model by which to enhance their clinical decision making.

The concepts of moist wound healing and the significance of clinical treatment decisions regarding wound care dressing options described herein are essential elements of your wound care arsenal. Other therapies, such as ES, HBOT, ultrasound, and growth factors, to name a few, are also important wound healing options available for

consideration. By using helpful practice points, tables, figures, and product algorithms, the clinician is guided through the milieu of product alternatives. Improving technology and evolving research into wound care dressings and modalities will continue to create new and challenging opportunities for all of us.

PATIENT SCENARIO

Clinical Data

A 36-year-old male presented for evaluation and treatment of frostbite injury 24 hours following cold exposure. He complained of pain and numbness in the involved digits of his right hand, primarily his index, middle, ring, and small fingers. Physical examination was remarkable for cool finger tips with a slight purple discoloration consistent with early tissue ischemia. There was no blistering or frank tissue necrosis. Capillary refill was diminished in the involved digits with slight numbness to light touch when compared to the left unaffected hand (Fig. 9-28). The patient was offered hyperbaric oxygen therapy (HBOT) (2.0 ATA for 90 minutes) daily for several days until his symptoms resolved. Near-infrared reflectance spectroscopic (NIRS) imaging was used to assess the immediate response of the injured tissue to hyperbaric oxygenation.

Figure 9-28. Frostbite injury. (Used with permission of J. Niezgoda.)

Case Discussion

Monitoring, assessing, and measuring the response to therapeutic intervention continue to be the Holy Grail for wound care practitioners. Historically, we have been forced to rely on direct visual observations of the wound characteristics correlated with crude measurements during a given visit coupled with digital photographs to provide a retrospective analysis over a period of time. This methodology does not provide rapid assessment that can be used to determine if an intervention is providing benefit or not on a more immediate basis. Newer technologies are emerging that may allow for point of care assessment and determinations as to the efficacy of a given treatment or the therapeutic value that is being delivered.

Near-infrared reflectance spectroscopic (NIRS) imaging can be used to visualize hemoglobin oxygen saturation in the vascular beds

just below the surface of tissue. Like pulse oximetry where tissue is illuminated with specific wavelengths of light in the red to near-infrared range to determine the proportion of hemoglobin carrying oxygen, near-infrared spectroscopy using those same illumination wavelengths can provide the hemoglobin oxygen saturation of an area of tissue (Fig. 9-28). However, unlike the S_pO_2 from pulse oximetry, which primarily measures the hemoglobin saturation in the larger arteries, the hemoglobin oxygen saturation provided by near-infrared reflectance spectroscopic imaging is a composite saturation from the entire vascular bed. This localized measure of the composite hemoglobin oxygen saturation has been dubbed tissue hemoglobin oxygen saturation, S_tO_2. This measure contrasts to transcutaneous oxygen pressure measurements ($TcPO_2$), which measures the free oxygen that has diffused to the surface of the tissue from the underlying vessels (Fig. 9-29). While $TcPO_2$ measurements require sensor probes to be applied to the tissue, S_tO_2 imaging is a rapid, noncontact, real-time measurement requiring little or no wound site preparation and no consumables. S_tO_2 is sensitive to local variations in hemoglobin oxygen delivery, utilization, and venous return as it surveys the extent of hemoglobin oxygen desaturation driven by local cellular respiration. S_tO_2 images can be used to detect and localize regional hemodynamic abnormalities in the vascular beds just below the surface of the tissue. In the context of vascular and wound assessment, adequate oxygen delivery and utilization is requisite for tissue viability.

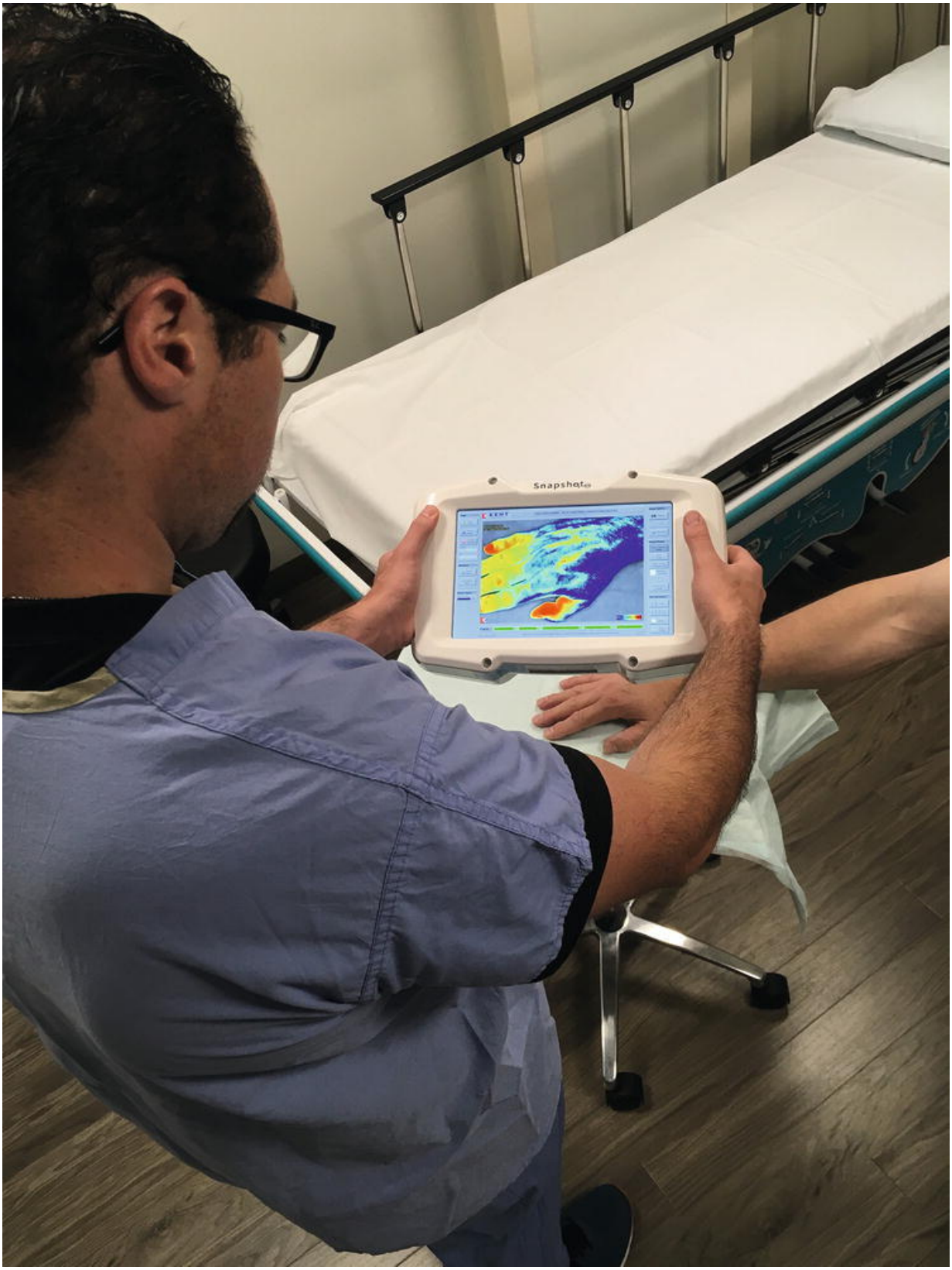
Snapshot

Figure 9-29. Equipment used to take scans. (Used with permission of J. Niezgoda.)

Figures 9-28 and 9-30 are the initial color digital image and near-infrared reflectance spectroscopic (NIRS) S_tO_2 image of the affected hand at the time of the initial consultation for frostbite injury (Figs. 9-28 and 9-30). NIRS imaging demonstrated the profound tissue injury and lack of tissue perfusion due to the frostbite. Figure 9-31 is an S_tO_2 image of the same hand on the same day but immediately following hyperbaric therapy. Figure 9-32 is an S_tO_2 image of the same hand 48 days after presentation; Figures 9-33 and 9-34 are the S_tO_2 image and color picture of the same hand 70 days after presentation. Serial NIRS imaging clearly highlights the utility of near-infrared reflectance S_tO_2 imaging for detecting local hemoglobin oxygenation deficits and monitoring the hemodynamic response to therapy. This technology correlated with an excellent patient outcome clearly demonstrated nearly instantaneously in real time the positive impact of HBOT on tissue perfusion and oxygenation.

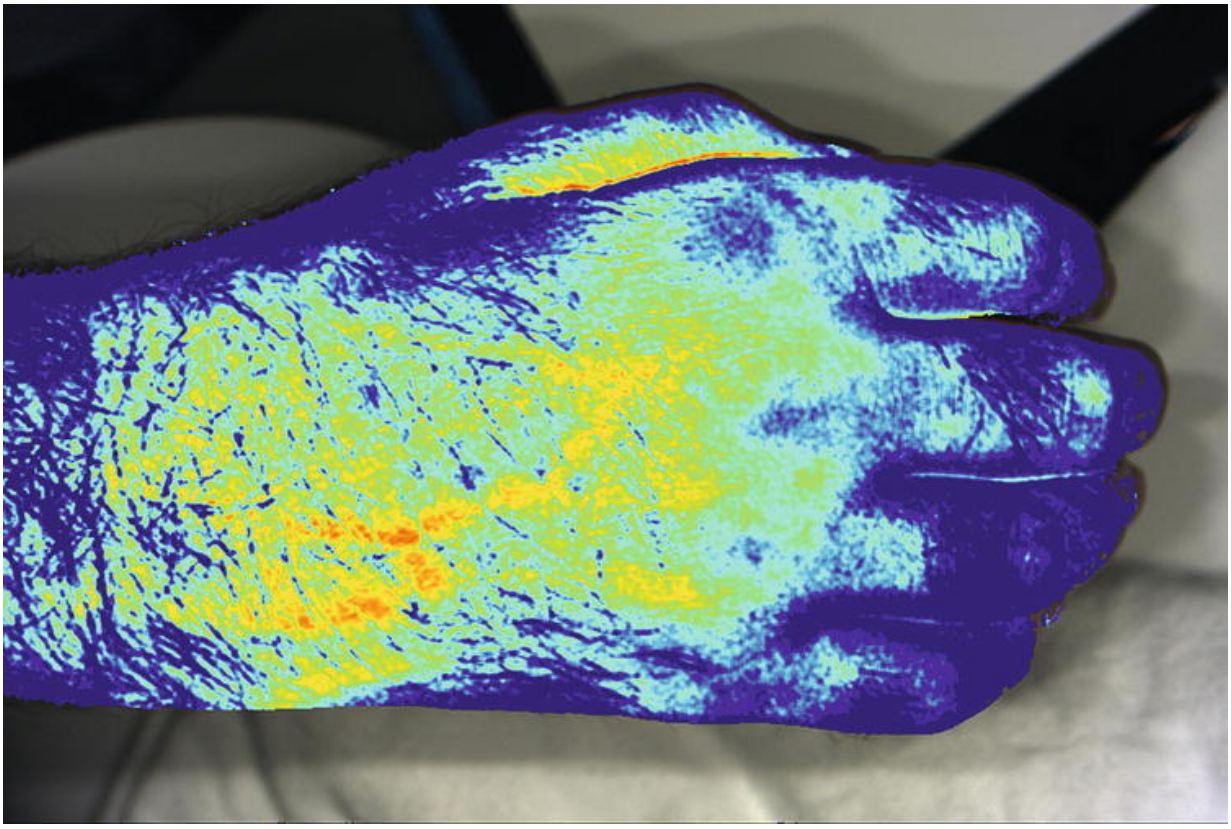

Figure 9-30. A near-infrared reflectance S_tO_2 image of the hand taken at the same time as the color picture in panel A. (Used with permission of J. Niezgoda.)

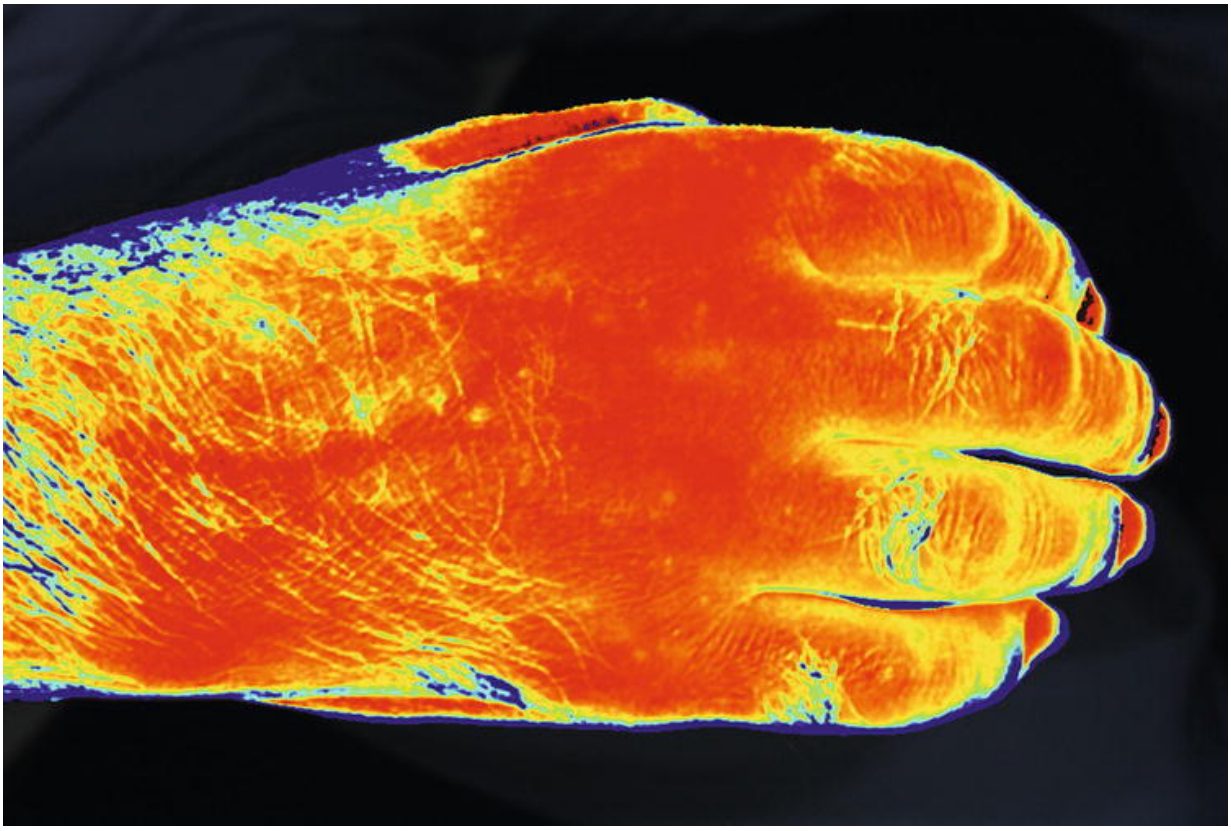

Figure 9-31. An S_tO_2 image of the same hand on the same day as A and B but immediately following hyperbaric therapy. (Used with permission of J. Niezgoda.)

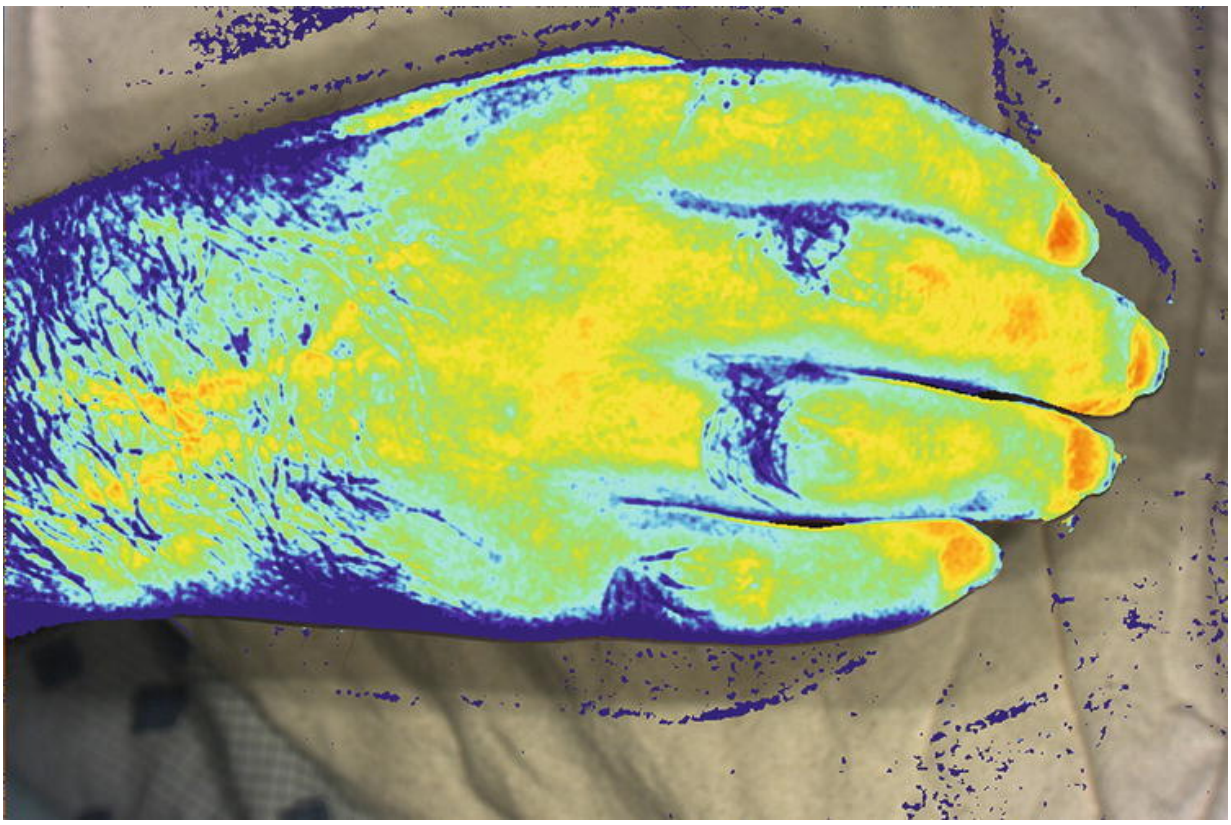

Figure 9-32. An S_tO_2 image of the same hand 48 days after presentation. (Used with permission of J. Niezgoda.)

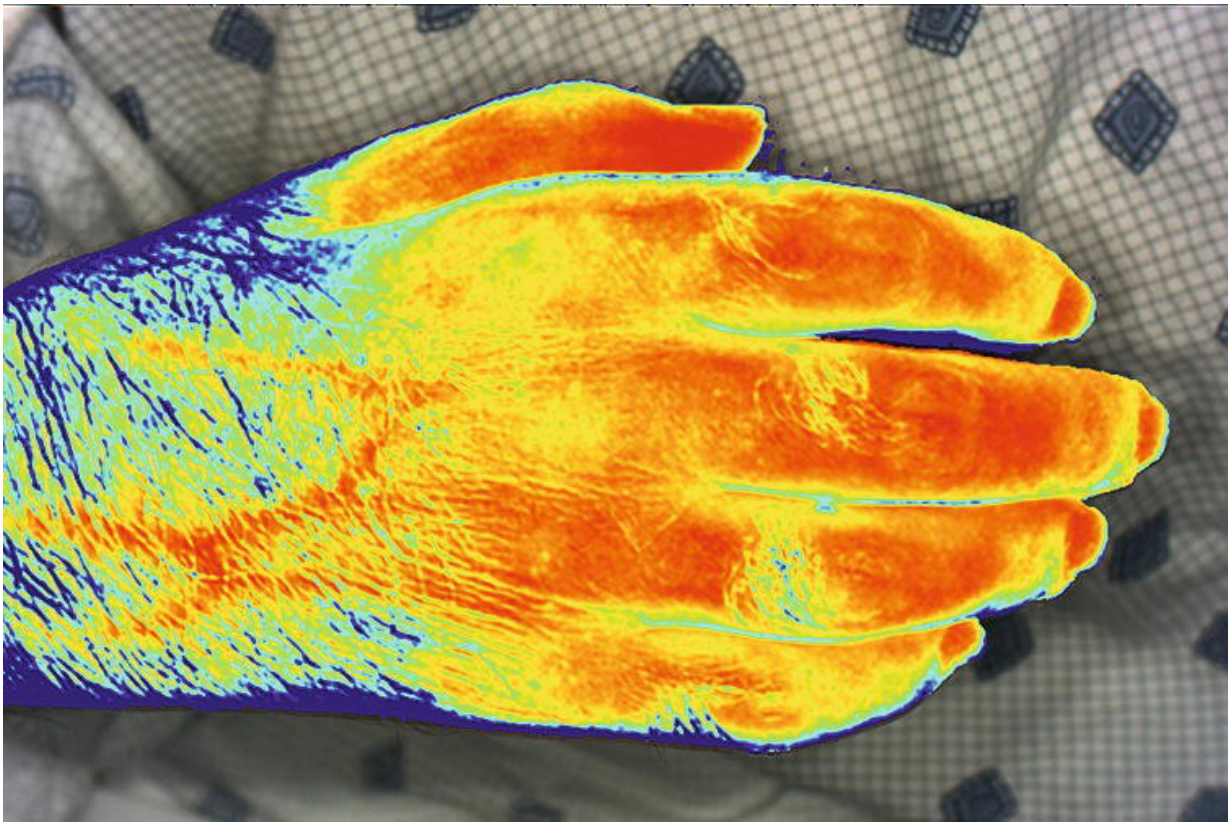

Figure 9-33. The S_tO_2 image and color picture of the same hand 70 days after presentation. (Used with permission of J. Niezgoda.)

Figure 9-34. The S_tO_2 image and color picture of the same hand 70 days after presentation. (Used with permission of J. Niezgoda.)

Show What You Know

1. **Wound dressings have evolved into a new concept of:**
   - A. dry gauze.
   - B. moist wound therapy.
   - C. open to air.
   - D. wet to dry.

2. **Which of the following is *not* a category of moist wound care dressings?**
   - A. Hydrogel dressing
   - B. Calcium alginate dressing
   - C. Roller gauze dressing
   - D. Foam dressing

3. **Wound dressing selection should be based on the characteristics of the wound. All of the following should be considered when selecting dressings *except*:**
   - A. size of dressing.
   - B. nurse preference.
   - C. moist or dry wound bed.
   - D. drainage.

4. **A disadvantage of transparent film is that it:**
   - A. is nonabsorptive.
   - B. is conformable.
   - C. allows wound inspection.
   - D. is impermeable to bacteria.

5. **The acronym "MEASURES" is a useful tool for remembering the principles of wound care.**
   - A. True
   - B. False

6. **Which one of the following is a dermal skin substitute?**
   - A. OPSITE

B. Tegaderm
C. Allevyn
D. Apligraf

7. **Which one of the following treatment options would *not* be an appropriate treatment option for a heavily draining wound?**
   A. Negative pressure therapy
   B. Foam dressing
   C. Calcium alginate dressing
   D. Hydrogel amorphous gel

8. **Prior to the application of tissue-engineered skin substitute, the clinician needs to evaluate:**
   A. whether the wound bed is free of necrotic tissue.
   B. the patient's immune system status.
   C. that antirejection drugs have been administered.
   D. the patient's weight.

9. **All of the following are therapeutic effects of electrical stimulation *except*:**
   A. increased blood flow.
   B. decreased tissue oxygenation.
   C. increased angiogenesis.
   D. decreased wound pain.

10. **Which one of the following is *not* a method by which hyperbaric oxygen therapy affects wound healing?**
   A. Increased fibroblast replication
   B. Up-regulation of growth factors
   C. Vasodilation effects of oxygen on the blood vessels
   D. Enhanced leukocyte bactericidal effects

Answers to these questions are found on page 683.

References

1. Ayello, E.A. “20 Years of Wound Care: Where We Have Been, Where We Are Going.” Advances in Skin & Wound Care 19(1):28-33, January-February 2006.
2. Queen, D. “A Personal Perspective.” International Wound Journal (1):1, March 2006.
3. Milne, C.T. Clinical Editor. “Wound Source 2014: The Kestrel Wound Product Sourcebook,” 17th ed. Hinesburg, VT: Kestrel Health Information, 2014.
4. Winter, G.D. “Formation of the Scab and the Rate of Epithelialization of Superficial Wounds in the Skin of Young Domestic Pigs.” Nature 193:293-4, 1962.
5. Hinman, C.D., Maibach, H.I. “Effect of Air Exposure and Occlusion on Experimental Human Skin Wounds.” Nature 200:377, 1963.
6. Baranoski, S. “Wound Dressings: An Evolving Art and Science.” Advances in Skin & Wound Care 25(2):87-92, February 2012.
7. Baranoski, S. “Wound & Skin Care: Choosing a Wound Dressing, Part 1.” Nursing 2008:60-1, January 2008.
8. Baranoski, S. “Wound & Skin Care: Choosing a Wound Dressing, Part 2.” Nursing 2008:14-5, February 2008.
9. Ovington, L. “Dressings and Skin Substitutes.” In J. McCulloch, L. Kloth, eds. Wound Healing Evidence-Based Management, 4th ed. Philadelphia, PA: F.A. Davis, 2010:183-6.
10. Armstrong, M.H., Price, P. “Wet to Dry Dressings: Fact and Fiction.” Available at: http://www.woundsresearch.com/article/2284. Accessed August 17, 2010.
11. Pieper, B., et al. “Wound Prevalence, Types, and Treatments in Home Care.” Advances in Skin & Wound Care 12(3):117-26, May-June 1999.
12. Kim, J.K., et al. “Normal Saline Wound Dressing—Is It Really Normal?” British Journal of Plastic Surgery 53(1):42-5, January 2000.
13. WoundPedia. Principles of Best Practice: Minimising Pain at Wound-Dressing-Related Procedures. A Consensus Document. Toronto, ON: WoundPedia, Inc., 2007.
14. Lawrence, J.C. “Dressings and Wound Infection.” American Journal of Surgery 167 (Suppl 1A):1S-24, January 1994.
15. Hutchinson, J.J. “Prevalence of Wound Infection under Occlusive Dressings: A Collective Survey of Reported Research.” Wounds (1):123-33, 1989.
16. Hutchinson, J.J. “A Prospective Clinical Trial of Wound Dressings to Investigate the Rate of Infection under Occlusion.” In K. Harding, ed. Proceedings of the First European Conference on Advances in Wound Management. London, UK: Macmillan, 1993.
17. Okan, D., Woo, K., Ayello, E.A., et al. “The Role of Moisture Balance in Wound Healing.” Advances in Skin & Wound Care 20:39-53, 2007.

18. Baranoski, S., McIntosh, A., Montoya, L. “Optimizing the Use of Dressings: What’s Best for my Patient?” Clinical Symposium on Advances in Skin & Wound Care, Las Vegas, NV, September 2014.
19. Seaman, S. “Dressing Selection in Chronic Wound Management.” Journal of the American Podiatric Medical Association 92(1):24-33, January 2002.
20. Doughty, D.B. “Wound Care in Long Term Care: Focus on Infectious Complications.” Safe Practices in Patient Care 4(3):15-7, 2010.
21. Rodeheaver, G. “Wound Cleansing, Wound Irrigation, Wound Disinfection.” In D. Krasner, G. Rodeheaver, R.G. Sibbals, eds. Chronic Wound Care, 4th ed. Malvern, PA: HMP Communications, 2007.
22. San Miguel, L., Torra I Bou, J.E., et al. “Economics of Pressure Ulcer Care: Review of the Literature on Modern Versus Traditional Dressings.” Journal of Wound Care 16(1):5-9, January 2007.
23. Payne, W.G., Posnett, J., et al. “A Prospective, Randomized Clinical Trial to Assess the Cost-Effectiveness of a Modern Foam Dressing versus a Traditional Saline Gauze Dressing in the Treatment of Stage II Pressure Ulcers.” Ostomy/Wound Management 55(2):50-5, February 2009.
24. Davidson, J. “Growth Factors and Extracellular Matrix in Wound Repair.” In J. Mculloch, L. Kloth, eds. Wound Healing Evidence-Based Management, 4th ed. Philadelphia, PA: F.A. Davis, 2010:35-43.
25. Orland, G. “The Fine Structure of the Interrelationship of Cells in the Human Epidermis.” Journal of Biophysical and Biochemical Cytology 4:529-35, 1958.
26. Eaglstein, W.H. “From Occlusive to Living Membranes.” Journal of Dermatology 25(12):766-74, December 1998.
27. Choucair, M., Phillips, T.J. “Wound Dressings.” In I.M. Freedberg, A.Z. Eisen, K. Wolff, et al, eds. Fitzpatrick’s Dermatology in General Medicine, 5th ed. New York: McGraw-Hill, 1999:2954-8.
28. Hess, C.T. Clinical Guide to Skin and Wound Care, 7th ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2013.
29. Hochstein, A.O., Bhatia, A. Collagen: Its Role in Wound Healing. Podiatry Management, August 2014:103-10.
30. Rangaraj, A., Harding, K.I., Leaper, D. “Role of Collagen in Wound Management.” Wounds 7(2):54-63, 2011.
31. Sussman, G. Management of the Wound Environment with Dressing and Topical Agents. In C. Sussman, B.M. Bates-Jensen, eds. Wound Care: A Collaborative Practice Manual for Health Professionals, 4th ed. Baltimore, MD: Lippincott Williams & Wilkins, 2012:502-21.
32. Niezgoda, J.A. Adjunctive Interventions for Limb Salvage & Wound Management: An Integrated Approach to Limb Salvage. Milwaukee, WI, September 2005. Webinar Streamed on http://WebCME.net
33. Sheehan, P., et al. “Percent Change in Wound Area of Diabetic Foot Ulcers Over a 4-Week Period Is a Robust Predictor of Complete Healing in a 12-Week Prospective Trial.” Diabetes Care 26:1879-82, 2003.

34. Snyder, R.J. “Consensus Recommendations on Advancing the Standard of Care for Treating Neuropathic Foot Ulcers in Patients with Diabetes.” Ostomy/Wound Management 56(4 Suppl):S1-24, April 2010. Available at: http://www.cms.gov/medicare-coverage-database/details/ncd-hyperbaricoxygentherapy. Accessed August 2014.
35. Niezgoda, J.A., Kindwall, E.P. “Chapter 32: Oxygen Therapy: The Management of the Hypoxic Wound.” In J.M. McCulloch, L. Kloth, eds. Wound Healing: Alternatives in Management, 4th ed., Philadelphia, PA: F. A. Davis, 2011.
36. Emily, H., ed.; National Pressure Ulcer Advisory Panel, European Pressure Ulcer Advisory Panel and Pan Pacific Pressure Injury Alliance. Prevention and Treatment of Pressure Ulcers: Clinical Practice Guideline. Osborne Park, Western Australia: Cambridge Media, 2014.
37. Agency for Healthcare Research and Quality. Negative Pressure Wound Therapy Devices. Available at: http://www.ahrq.gov/clinic/ta/negpresswtd/npwtd02.htm. Accessed September 2, 2010.
38. Paul, J.C. “Vacuum Assisted Closure Therapy: A Must in Plastic Surgery.” Plastic Surgical Nursing 25(2):61-5, April-June 2005.
39. Gupta, S., Gabriel, A., Niezgoda, J.A. “Differentiating Negative Pressure Wound Therapy Devices: An Illustrative Case Series.” Wounds 19(1 Suppl):1-9, 2007.
40. Kilpadi, D.V., Stechmiller, J.K., Childress, B., et al. “Composition of Wound Fluid from Pressure Ulcers Treated with Negative Pressure Wound Therapy Using V.A.C.® Therapy in Home Health.” Wounds 18(5):119-26, 2006.
41. Willy, C., ed. The Theory and Practice of Vacuum Therapy. Scientific Basis, Indications for Use, Case Reports, Practical Advice. Ulm, Germany: Lindqvist Book Publishing, 2006:405.
42. Borgquist, O., Ingemansson, R., Malmsjo, M. “Negative Pressure Wound Therapy Using Gauze and Foam: An In-detail Study of the Effects on the Wound Bed Including Macro and Microdeformation, Tissue Ingrowth and Wound Bed Histology.” Department of Ophthalmology and Department of Cardiothoracic Surgery, Lund University Hospital, Lund, Sweden. Presented at the 24th Annual Clinical Symposium on Advances in Skin & Wound Care in San Antonio, TX, October 22–25, 2009.
43. Weed, T., Ratliff, C., Drake, D.B. “Quantifying Bacterial Bioburden During Negative Pressure Wound Therapy: Does the Wound VAC Enhance Bacterial Clearance?” Annals of Plastic Surgery 52(3):276-9, discussion 279-80, March 2004.
44. Moues, C.M., Vos, M.C., van den Bemd, G.J., et al. “Bacterial Load in Relation to Vacuum-assisted Closure Wound Therapy: A Prospective Randomized Trial.” Wound Repair and Regeneration 12(1):11-7, January-February 2004.

45. Armstrong, D.G., Marston, W.A., Reyzelman, A.M., et al. “Comparative Effectiveness of Mechanically and Electrically Powered Negative Pressure Wound Therapy Devices: A Multicenter Randomized Controlled Trial.” Wound Repair and Regeneration 20(3):332-41, 2012.
46. Shum, D., Suzuki, K. “Emerging Insights on Negative Pressure Wound Therapy.” Podiatry Today 26: 50-7, August 2013.
47. Hutton, D.W., Sheehan, P. “Comparative Effectiveness of the SNaP™ Wound Care System.” International Wound Journal 8(2):196-205, April 2011.
48. Saxena, V., Hwang, C.W., Huang, S., et al. “Vacuum-assisted Closure: Microdeformations of Wounds and Cell Proliferation.” Plastic and Reconstructive Surgery 114(5):1086-1096, discussion 1097, October 2004.
49. Scherer, S.S., Pietramaggiori, G., Mathews, J.C., et al. “The Mechanism of Action of the Vacuum-assisted Closure Device.” Plastic and Reconstructive Surgery 122(3):786-1097, September 2008.
50. Malmsjo, M., Ingemansson, R., Martin, R., et al. “Negative-pressure Wound Therapy using Gauze or Open Cell Polyurethane Foam: Similar Early Effects on Pressure Transduction and Tissue Contraction in an Experimental Porcine Wound Model.” Wound Repair and Regeneration 17(2):200-5, March-April 2009.
51. Morykwas, M.J., Faler, B.J., Pearce, D.J., et al. “Effects of Varying Levels of Subatmospheric Pressure on the Rate of Granulation Tissue Formation in Experimental Wounds in Swine.” Annals of Plastic Surgery 47(5):547-51, November 2001.
52. Niezgoda, J.A. “Combining Negative Pressure Wound Therapy with Other Wound Management Modalities.” Ostomy/Wound Management 51(2A Suppl):36S-8, February 2005.
53. Niezgoda, J.A., Sordi, P.J., Hermans, M.H.E. “Evaluation of Vashe Wound Therapy in the Clinical Management of Patients with Chronic Wounds.” Advances in Skin & Wound Care 23(8), 352-57, August 2010.
54. Hettrick, H. “Lymphedema Complicating Healing.” In J.M. McCulloch, L.C. Kloth, eds. Wound Healing: Evidence-Based Management, 4th ed. Philadelphia, PA: F.A. Davis, 2010:279-91.
55. Rabe, E., Partsch, H., Hafner, J., et al. Indications for Medical Compression Stockings in Venous and Lymphatic Disorders: An Evidence-Based Consensus Statement. Phlebology 33(3):163-84, 2017.
56. McCulloch, J.M. “Compression Therapy.” In J.M. McCulloch, L.C. Kloth, eds. Wound Healing: Evidence-Based Management, 4th ed. Philadelphia, PA: F.A. Davis, 2010:594-601.
57. Partsch, H., Mexzinger, G., Mostobeck, A. Inelastic Leg Compression Is More Effective to Reduce Deep Venous Reflux Than Elastic Bandages. Dermatologic Surgery 25:695-700, 1999.
58. Ayello, E.A., Baranoski, S. “2014 Survey Results: Wound Care and Prevention.” Advances in Skin & Wound Care 27(8):371-80, 2014, quiz 381-2.

59. Smart, H. “The Africa Bandage System and Application Technique-Bringing Compression Science to Resource-Restrained Environment.” World Council of Enterostomal Therapists Journal 34(2):8-16, 2014.
60. Dai, G.M.S., et al. “An In Vitro Cell Culture System to Study the Influence of External Pneumatic Compression on Endothelial Function.” Journal of Vascular Surgery 32:977-87, 2000.
61. Kessler, C.M., et al. “Intermittent Pneumatic Compression in Chronic Venous Insufficiency Favorably Affects Fibrinolytic Potential and Platelet Activation.” Blood Coagulation and Fibrinolysis 7:437, 1996.
62. Alpagut, U., Dayioglu, E. “Importance and Advantages of Intermittent External Pneumatic Compression Therapy in Venous Stasis Ulceration.” Angiology 56(1):19-23, 2005.
63. Tran, K., Argáez, C. *Intermittent Pneumatic Compression Devices for the Management of Lymphedema: A Review of Clinical Effectiveness and Guidelines* [Internet]. Ottawa, ON: Canadian Agency for Drugs and Technologies in Health, 2017 May 12. Conclusions and Implications for Decision or Policy Making.
64. Alvarez, O., Wendelken, M., Markowits, L., et al. Effect of High-Pressure, Intermittent Pneumatic Compression for the Treatment of Peripheral Arterial Disease and Critical Lung Ischemia in Patients With a Surgical Option. Wounds 27(11):293-301, November 2015.
65. Delis, K.T., Nicolaides, A.N., Wolfe, J.H., et al. “Improving Walking Ability and Ankle Brachial Pressure Indices in Symptomatic Peripheral Vascular Disease with Intermittent Pneumatic Foot Compression: A Prospective Controlled Study with One-Year Follow-up.” Journal of Vascular Surgery 31:650-61, 2000.
66. Van Bremmelen, P.S., Gitlitz, D.B., Faruqi, R.M. “Limb Salvage Using High-Pressure Intermittent Compression Arterial Assist Device in Cases Unsuitable for Surgical Revascularization.” Archives of Surgery 136:1280-5, 2001.
67. Kindwall, E.P., Niezgoda, J.A., eds. Hyperbaric Medicine Procedures: The Kindwall HBO Handbook. Milwaukee, WI: Aurora Heath Care, 2006.
68. Piccolino, M. “Animal Electricity and the Birth of Electrophysiology. The Legacy of Luigi Galvani.” Brain Research Bulletin 46:381-407, 1998.
69. Piccolino, M. “Luigi Galvani’s Path to Animal Electricity.” Comptes Rendus Biology 329:303-18, 2006.
70. Moruzzi, G. “The Electrophysiological Work of Carlo Matteucci.” Brain Research Bulletin 40:69-91, 1996.
71. Kloth, L., Zhao, M. “Endogenous and Exogenous Electrical Fields for Wound Healing.” In J. McCulloch, L. Kloth, eds. Wound Healing Evidence-Based Management, 4th ed. Philadelphia, PA: F.A. Davis, 2010:450-513.
72. Hunckler, J., de Mel, A. A Current Affair: Electrotherapy in Wound Healing. Journal of Multidisciplinary Healthcare 10:179-94, 2017. doi: 10.2147/JMDH.S127207.

73. Kloth, L.C. “5 Questions and Answers about Electrical Stimulation.” Advances in Skin & Wound Care 14(3):156-8, May-June 2001.
74. Garber, S.L., et al. Pressure Ulcer Prevention and Treatment Following Spinal Cord Injury: A Clinical Practice Guideline for Health-care Professionals, Consortium for Spinal Cord Medicine Clinical Practice Guidelines. Washington, DC: Paralyzed Veterans of America, 2014.
75. Junger, M., et al. “Treatment of Venous Ulcers with Low Frequency Pulsed Current (Dermapulse): Effects on Cutaneous Microcirculation.” Der Hautartzt 18:879-903, 1997.
76. Gagnier, K.A., et al. “The Effects of Electrical Stimulation on Cutaneous Oxygen Supply in Paraplegics.” Physical Therapy 68:835, 1988.
77. Dodgen, P.W., et al. “The Effects of Electrical Stimulation on Cutaneous Oxygen Supply in Paraplegics.” Physical Therapy 67:793, 1987.
78. Greenberg, J., et al. “The Effect of Electrical Stimulation (RPES) on Wound Healing and Angiogenesis in Second-degree Burns.” Abstract # 44 in Program and Abstracts of the 13th Annual Symposium on Advanced Wound Care, Dallas, TX, April 1–4, 2000.
79. Kloth, L.C., McCulloch, J.M. Wound Healing Alternatives in Management, 3rd ed. Philadelphia, PA: F.A. Davis, 2002:271-315.
80. Brown, M., et al. “Electrical Stimulation Effects on Cutaneous Wound Healing in Rabbits.” Physical Therapy 68:955, 1988.
81. Demir, H., et al. “A Comparative Study of the Effects of Electrical Stimulation and Laser Treatment on Experimental Wound Healing in Rats.” Journal of Rehabilitation Research & Development 41(2):147-54, March 2004.
83. Myer, A. “The Role of Physical Therapy in Chronic Wound Care.” In D. Krasner, et al., eds. Chronic Wound Care: A Clinical Source Book for Healthcare Professionals, 3rd ed. Wayne, PA: Health Management Publications, Inc., 2001.
84. Tai, G., Tai, M., Zhao, M. Electrically Stimulated Cell Migration and Its Contribution to Wound Healing. Burns & Trauma 6:20, July 9 2018.
82. Houghton, P.E., Campbell, K.E. “Therapeutic Modalities in the Treatment of Chronic Recalcitrant Wounds.” In D. Krasner, et al., eds. Chronic Wound Care: A Clinical Source Book for Healthcare Professionals, 3rd ed. Wayne, PA: Health Management Publications, Inc., 2001:455-68.
85. Polak, A., et al. A randomized, Controlled Clinical Study to Assess the Effect of Anodal and Cathodal Electrical Stimulation on Periwound Skin Blood Flow and Pressure Ulcer Size Reduction in Persons with Neurological Injuries. Ostomy Wound Management 64(2):10-29, February 2018.
86. Conner-Kerr, T. “Light Therapies.” In J. McCulloch, L. Kloth, eds. Wound Healing Evidence-Based Management, 4th ed. Philadelphia, PA: F.A. Davis, 2010:576-93.
87. Steinlechner, C., Dyson, M. “The Effect of Low Level Laser Therapy on the Proliferation of Keratinocytes.” Laser Therapy 5(2):65, 1993.

88. Dyson, M., Young, S. “Effect of Laser Therapy on Wound Contraction and Cellularity in Mice.” Lasers in Medical Science 1:125, 1986.
89. Dyson, M. “Cellular and Subcellular Aspects of Low Level Laser Therapy.” In T. Ohshiro, R.G. Calderhead, eds. Progress in Laser Therapy. London, UK: John Wiley & Sons, 1991:221.
90. Bolton, P.A., et al. “Macrophage Responsiveness to Light Therapy. A Dose Response Study.” Laser Therapy 2:101-6, 1990.
91. Bolton, P.A., et al. “The Effect of Polarised Light on the Release of Growth Factors from the U-937 Macrophage-like Cell Line.” Laser Therapy 4:33-42, 1992.
92. Cheetham, M.J., et al. “Histological Effects of 820 nm Laser Irradiation on the Healthy Growth Plate of the Rat.” Laser Therapy 2:59, 1992.
93. Bolton, P.A., et al. “The Direct Effect of 860 nm Light on Cell Proliferation and on Succinic Dehydrogenase Activity of Human Fibroblasts In vitro.” Laser Therapy 7:55-60, 1995.
94. el Sayed, S.O., Dyson, M. “Effect of Laser Pulse Repetition Rate and Pulse Duration on Mast Cell Number and Degranulation.” Lasers in Surgery and Medicine 19(4):433-7, 1997.
95. Kazemikhoo, N., Vaghardoost, R., Dahmardehei, M., et al. Evaluation of the Effects of Low Level Laser Therapy on the Healing Process After Skin Graft Surgery in Burned Patients (A Randomized Clinical Trial). Journal of Lasers in Medical Sciences 9(2):139-43, 2018.
96. Taradaj, J., Shay, B., Dymarek, R., et al. Effect of Laser Therapy on Expression of Angio- and Fibrogenic Factors, and Cytokine Concentrations During the Healing Process of Human Pressure Ulcers. International Journal of Medical Sciences 15(11):1105-12, 2018.
97. Nicolopoulos, N., et al. “The Use of Laser Surgery in the Subtotal Meniscectomy and the Effect of Low-level Laser Therapy on the Healing Potential of Rabbit Meniscus: An Experimental Study.” Lasers in Medical Science 1(2):109-15, 1996.
98. Crous, L.C., Malherbe, C.P. “Laser and Ultraviolet Light Irradiation in the Treatment of Chronic Ulcers.” South African Journal of Physiotherapy 44(3):73-7, 1988.
99. Franek, A., et al. “Does Low Output Laser Stimulation Enhance the Healing of Crural Ulceration? Some Critical Remarks.” Medical Engineering and Physics 24(9):607-15, 2002.
100. Iusim, M., et al. “Evaluation of the Degree of Effectiveness of Biobeam Low Level Narrow Band Light on the Treatment of Skin Ulcers and Delayed Postoperative Wound Healing.” Orthopedics 15(9): 1023-6, 1992.
101. Lagan, K.M., et al. “Low-intensity Laser Therapy/Combined Phototherapy in the Management of Chronic Venous Ulceration: A Placebo-controlled Study.” Journal of Clinical Laser Medicine and Surgery 20(3):109-16, 2002.
102. Lucas, C., et al. “The Effect of Low Level Laser Therapy (LLLT) on Stage III Decubitus Ulcers (Pressure Sores); A Prospective Randomised Single Blind,

Multicentre Pilot Study." Lasers in Medical Science 15(2):94-100, 2000.
103. Lucas, C., et al. "Efficacy of Low-level Laser Therapy in the Management of Stage III Decubitus Ulcers: A Prospective, Observer-blinded Multicentre Randomised Clinical Trial." Lasers in Medical Science 18(2):72-7, 2003.
104. Lundeberg, T., Malm, M. "Low-power HeNe Laser Treatment of Venous Leg Ulcers." Annals of Plastic Surgery 27(6):537-9, 1991.
105. Malm, M., Lundeberg, T. "Effect of Low Power Gallium Arsenide Laser on Healing of Venous Ulcers." Scandinavian Journal of Plastic Reconstructive Surgery and Hand Surgery 25(3):249-51, 1991.
106. Nussbaum, E.L., et al. "Comparison of Ultrasound/Ultraviolet-C and Laser for Treatment of Pressure Ulcers in Patients with Spinal Cord Injury." Physical Therapy 74(9):812-23, 1994.
107. Kloth, L.C., McCulloch, J.M. Wound Healing Alternatives in Management, 3rd ed. Philadelphia, PA: F.A. Davis, 2002:326-39.
108. Conner-Kerr, T., et al. "UVC Reduces Antibiotic-resistant Bacteria In vitro." Ostomy/Wound Management 45:84, 1999.
109. Thai, T., et al. "Effect of Ultraviolet Light C on Bacterial Colonization in Chronic Wounds." Ostomy/Wound Management 51(10):32-45, 2005.
110. Thai, T., et al. "Ultraviolet Light C in the Treatment of Chronic Wounds with MRSA: A Case Study." Ostomy/Wound Management 48(11):52-60, 2002.
111. Conner-Kerr, T., et al. "The Effects of Ultraviolet Irradiation on Antibiotic-resistant Bacteria In vitro." Ostomy/Wound Management 44:508-11, 1998.
112. Kloth, L.C., McCulloch, J.M. Wound Healing Alternatives in Management, 3rd ed. Philadelphia, PA: F.A. Davis, 2002:335.
113. Gupta, A., Avci, P., Dai, T., et al. Ultraviolet Radiation in Wound Care: Sterilization and Stimulation. Advances in Wound Care 2(8):422-37, 2013.
114. Ennis, W.J., et al. "Ultrasound Therapy for Recalcitrant Diabetic Foot Ulcers: Results of a Randomized, Double-blind, Controlled, Multicenter Study." Ostomy/Wound Management 51(8):24-6, 28-9, 32-9, August 2005.
115. McDiarmid, T., Ziskin, M.C., Michlovitz, S.L. "Therapeutic Ultrasound." In S.L. Michlovitx, ed. Thermal Agents in Rehabilitation, 3rd ed. Philadelphia, PA: F.A. Davis Company, 1996:168-212.
116. Kavros, S.J., Coronado, R. Diagnostic and Therapeutic Ultrasound on Venous and Arterial Ulcers: A Focused Review. Advances in Skin & Wound Care 31(2):55-65, 2018.
117. Stanisic, M.M., et al. "Wound Debridement with 25 kHz Ultrasound." Advances in Skin & Wound Care 18(9):484-90, November-December 2005.
118. Kloth, L., Niezgoda, J.A. "Chapter 28: Ultrasound for Wound Bed Preparation and Healing." In J.M. McCulloch, L. Kloth, eds. Wound Healing: Alternatives in Management, 4th ed. Philadelphia, PA: F. A. Davis, 2011.
119. Watson, T. Available at: http://www.electrotherapy.org/electro/ultrasound/therapeutic_ultrasound.htm. Accessed August 12, 2014.

120. Dyson, M. “Therapeutic Applications of Ultrasound.” In W.L. Nyborg, M.C. Ziskin, eds. Biological Effects of Ultrasound. Clinics in Diagnostic Ultrasound. New York: Churchill Livingstone, 1985:121-33.
121. Harvey, W., Dyson, M., Pond, J.B., et al. “The Stimulation of Protein Synthesis in Human Fibroblasts by Therapeutic Ultrasound.” Rheumatology and Rehabilitation 14(4):237-41, 1975.
122. Ennis, W.J., et al. “Evaluation of Clinical Effectiveness of MIST Ultrasound Therapy for the Healing of Chronic Wounds.” Advances in Skin & Wound Care 19(8):437-46, October 2006.
123. Kloth, L.C., Niezgoda, J.A. “Ultrasound for Wound Debridement and Healing.” In J.M. McCulloch, L.C. Kloth, eds. Wound Healing: Evidence-Based Management, 4th ed. Philadelphia, PA: F.A. Davis, 2010:545-75.
124. Honaker, J.S., Forston, M.R., Davis, E.A., et al. “Effects of Noncontact Low-Frequency Ultrasound on Healing of Suspected Deep Tissue Injury: A Retrospective Analysis.” International Wound Journal 10(1):65-72, 2013.
125. Available at: http://www.cms.gov/medicare_coverage_daabase/details/ned-hyperbaricoxygentherapy. Accessed August 22, 2014.
126. Available at: http://www.woundcarestakeholders.org/. Accessed August 22, 2014.
127. Hess, C.T., ed. “Advanced BioHealing Will Offer Dermagraft, TransCyte.” Advances in Skin & Wound Care 19(7):348, September 2006.
128. Dermagraft®. Available at: www.fda.gov/MedicalDevices/ProductsandMedicalProcedures/DeviceApprovalsandClearances/Recently-ApprovedDevices/ucm085085.htm. Accessed January 3, 2011.
129. Falanga, V., Margolis, D., Alvarez, O., et al. “Rapid Healing of Venous Ulcers and Lack of Clinical Rejection with an Allogeneic Cultured Human Skin Equivalent. Human Skin Equivalent Investigators Group.” Archives of Dermatology 134:293-300, 1998.
130. Marston, W., et al. “The Efficacy and Safety of Dermagraft in Improving the Healing of Chronic Diabetic Foot Ulcers.” Diabetes Care 26(6):1701-5, June 2003.
131. Tao, H., Fan, H. “Implantation of Amniotic Membrane to Reduce Postlaminectomy Epidural Adhesion.” European Spine Journal 18(8):1202-12, August 2009.
132. Koob, T., Rennert, R., Zabek, N., et al. “Biological Properties of Dehydrated Human Amnion/Chorion Composite Graft: Implications For Chronic Wound Healing.” International Wound Journal 10(5): 493-500, 2013.
133. Zelen, C., Serena, T., Denoziere, G., et al. “A Prospective Randomized Comparative Parallel Study on Amniotic Membrane Wound Graft in The Management of Diabetic Foot Ulcers.” International Wound Journal 10(5):502-07, 2013.
134. Kiistala, I. “Suction Blister Device for Separation of Viable Dermis.” Journal of Investigative Dermatology 50(2):129-37, 1968.

135. Demling, R.H., Niezgoda, J.A., Haraway, G.D., et al. “Small Intestinal Submucosa Wound Matrix and Full-thickness Venous Ulcers: Preliminary Results.” Wounds 16:18-22, January 2004.
136. Rader, R.A. “(Re)defining Biopharmaceutical.” Nature Biotechnology 26(7):743-51, 2008.
137. Available at: http://www.regranex.com/important-safety-info.php. Accessed June 2014.
138. Frykberg, R., Driver, V.R., et al. “Chronic Wounds Treated with a Physiologically Relevant Concentration of Platelet-rich Plasma Gel: A Prospective Case Series.” Ostomy/Wound Management 56(6):36-44, 2010.
139. McAleer, J.P., et al. “Use of Autologous Platelet Concentrate in a Nonhealing Lower Extremity Wound.” Advances in Skin & Wound Care 19(7):354-62, September 2006.
140. Prakash, J. “The Effect of Autologous Bone Marrow-Derived Cells on Healing Chronic Lower Extremity Wounds: Results of a Randomized Controlled Study.” Ostomy/Wound Management 57(7):38-42, 2011.
141. Thom, S.R., Veena, M. “Stem Cell Mobilization by Hyperbaric Oxygen.” American Journal of Physiology Heart and Circulatory Physiology 290: H1378-86, 2006.
142. Hammarlund, C. “The Physiologic Effects of Hyperbaric Oxygenation.” In E.P. Kindwall, H.T. Whelan, eds. Hyperbaric Medicine Practice. Flagstaff, AZ: Best Publishing, 2004:37-68.
143. Weaver, L.K., Valentine, K.J., Hopkins, R.O. “Carbon Monoxide Poisoning: Risk Factors for Cognitive Sequelae and the Role of Hyperbaric Oxygen.” American Journal of Respiratory and Critical Care Medicine 176:491-7, 2007.
144. Krough, A. “The Number and Distribution of Capillaries in Muscle with Calculations of the Oxygen Pressure Head Necessary for Supplying the Tissue.” Journal of Physiology 52:409-15, 1919.
145. Fife, C. “Hyperbaric Oxygen Therapy Applications in Wound Care.” In P. Sheffield, et al., eds. Wound Care Practice. Flagstaff, AZ: Best Publishing, 2004:664.
146. Available at: http://www.worldwidewounds.com/2001/april/Wright/HyperbaricOxygen.html.
147. Knighton, D., et al. “Regulation of Wound Healing Angiogenesis-effect of Oxygen Gradients and Inspired Oxygen Concentration.” Surgery 90: 262-70, 1981.
148. Sheikh, A.Y., Gibson, J.J., Rollins, M.D. “Effect of Hyperoxia on Vascular Endothelial Growth Factor Levels in a Wound Model.” Archives of Surgery 135(11):1293-7, November 2000.
149. Ahmad, Y., Sheikh, M.D., Mark, D. “Hyperoxia Improves Microvascular Perfusion in a Murine Wound Model.” Wound Repair and Regeneration 13(3):303-8, 2005.
150. Wright, J. “Hyperbaric Oxygen Therapy for Wound Healing.” World Wide Wounds, May 2001.

151. Bonomo, S.R., et al. “Hyperbaric Oxygen as a Signal Transducer: Upregulation of Platelet Derived Growth Factor-beta Receptor in the Presence of HBO2 and PDGF.” Undersea Hyperbaric Medicine 25(4):211-16, 1998.
152. Wu, L., et al. “Effects of Oxygen on Wound Responses to Growth Factors: Kaposi’s FGF, But not Basic FGF Stimulates Repair in Ischemic Wounds.” Growth Factors 12(1):29-35, 1995.
153. Knighton, D.R., et al. “Oxygen as an Antibiotic. The Effect of Inspired Oxygen on Infection.” Archives in Surgery 119(2):199-204, 1984.
154. Nylander, G., Nordström, H., Franzén, L. “Effects of Hyperbaric Oxygen Treatment in Post-ischemic Muscle A Quantitative Morphological Study.” Scandinavian Journal of Plastic and Reconstructive Surgery and Hand Surgery 22(1):31-9, 1988.
155. Buras, J. “Basic Mechanisms of Hyperbaric Oxygen in the Treatment of Ischemia-Reperfusion Injury.” International Anesthesiology Clinics 2000;38(1):91-109.
156. Available at: http://www.achm.org. Accessed August 22, 2014.
157. Available at: http://www.uhms.org. Accessed August 22, 2014.
158. Gray, M., Ratliff, C.R. “Is Hyperbaric Oxygen Therapy Effective for the Management of Chronic Wounds?” Journal of Wound, Ostomy, and Continence Nursing 33:21-5, 2006.
159. Gottrup, F., et al. “The Dynamic Properties of Tissue Oxygen in Healing Flaps.” Surgery 95(5):527-36, 1984.
160. Serena, T.E., Gelly, H., Bohn, G.A., et al. “The American College of Hyperbaric Medicine Consensus Statement on Physician Credentialing for Hyperbaric Oxygen Therapy.” Advances in Skin & Wound Care 27(8):349-51, August 2014.
161. Fries, R.B. “Dermal Excisional Wound Healing in Pigs Following Treatment with Topically Applied Pure Oxygen.” Mutation Research 579(1-2):172-81, 2005.
162. Lairet, K.F., Baer, D., Leas, M.L. “Evaluation of an Oxygen-Diffusion Dressing for Accelerated Healing of Donor-Site Wounds.” Journal of Burn Care & Research 35(3):214-18, May-June 2014.
163. Widgerow, A.D., Chait, L.A., Stahls, R., et al. “New Innovations in Scar Management.” Aesthetic Plastic Surgery 24:227-34, 2000.
164. Widgerow, A.D., Chait, L.A.C., Stahls, R., et al. “Multimodality Scar Management Program.” Aesthetic Plastic Surgery 33(4):533, 2009.
165. Widgerow, A.D. “Scar Management: The Principles and Their Practical Application.” World Council of Enterostomal Therapists Journal 31(1):18-21, 2011.
166. Chan, K.Y., et al. “Silicone Gel in Prevention of Hypertrophic Scar Development in Median Sternotomy Wound.” Plastic and Reconstructive Surgery 116:1013-20, 2005.

167. Elliot, D., Cory-Pearce, R., Rees, G.M. “The Behaviour of Presternal Scars in a Fair-skinned Population.” Annals of the Royal College of Surgery of England 67:238, 1985.
168. Meyer, M., McGrouther, D.A. “A Study Relating Wound Tension to Scar Morphology in the Presternal Scar Using Langers Technique.” British Journal Plastic Surgery 44:291, 1991.
169. Reiffel, R.S. “Prevention of Hypertrophic Scars by Long-term Paper Tape Application.” Plastic and Reconstructive Surgery 96:1715, 1995.
170. Atkinson, J.A., et al. “A Randomized Controlled Trial to Determine the Efficacy of Paper Tape in Preventing Hypertrophic Scar Formation in Surgical Excisions That Travers Langers Skin Tension Lines.” Plastic and Reconstructive Surgery 116(6):1648-54, November 2005.
171. Beauchamp, K., et al. “Phytochemistry: Ibuprofen-like Activity in Extra-virgin Olive Oil.” Nature 437:45-6, September 2005.
172. de la Puerta, R., Martinez-Dominguez, E., et al. “Effect of Minor Components of Virgin Olive Oil on Topical Anti-inflammatory Assays.” Verlag der Zeitschrift für Naturforschung 55(9-10):814, September-October 2000.
173. Katsiki, M., Chondrogianni, N., Chinou, I., et al. “The Olive Constituent Oleuropein Exhibits Proteasome Stimulatory Properties In vitro and Confers Life Span Extension of Human Embryonic Fibroblasts.” Rejuvenation Research 10(2):157-72, 2007.
174. Sawada, Y., Sone, K. “Hydration and Occlusion Treatment for Hypertrophic Scars and Keloids.” British Journal of Plastic Surgery 45:599, 1992.
175. Mustoe, T.A., et al. “International Clinical Recommendations on Scar Management [Review].” Plastic & Reconstructive Surgery 110(2):560-71, August 2002.
176. Sawada, Y., Urushidate, S., Nihei, Y. “Hydration and Occlusive Treatment of a Sutured Wound.” Annals of Plastic Surgery 41:508, 1998.
177. Niessen, F., et al. “The Use of Silicone Occlusive Sheet (Sil-K) and Silicone Occlusive Gel (Epiderm) in the Prevention of Hypertrophic Scar Formation.” Plastic & Reconstructive Surgery 102(6):1962-672, 1998.
178. Mustoe, T.A. “Evolution of Silicone Therapy and Mechanism of Action in Scar Management.” Aesthetic Plastic Surgery 32(3):82-92, March 2008.
179. Mustoe, T.A. “The Role of the Epidermis in the Control of Scarring: Evidence for Mechanism of Action for Silicone Gel.” Journal of Plastic, Reconstructive & Aesthetic Surgery 61(10):1219-25, October 2008.
180. Maquart, F.X., Bellon, G., Gillery, P., et al. “Stimulation of Collagen Synthesis in Fibroblast Cultures by a Triterpene Extracted from *Centella asiatica*.” Connective Tissue Research 24(2):107-20, 1990.
181. Zhang, T., Tian-zeng, L., Ying Bin, X. “Asiaticoside on Hypertrophic Scars of Transforming Growth Factor-β mRNA and Matrix Metalloproteinase Expression.” Journal of Southern Medical University 26(1):67-70, January 2006.

182. Bonte, F., Dumas, M., Chaudagne, C., et al. “Influence of Asiatic Acid, Madecassic Acid, and Asiaticoside on Human Collagen I Synthesis.” Planta Medica 60(2):133, April 1994.
183. Ju-Lin, X., Shao-Hai, Q., Tian-Zeng, L., et al. “Effect of Asiaticoside on Hypertrophic Scar in the Rabbit Ear Model.” Journal of Cutaneous Pathology 36(2):234-9, February 2009.
184. Saulis, A.S., Mogford, J.H., Mustoe, T.A. “Effect of Mederma on Hypertrophic Scarring in the Rabbit Ear Model.” Plastic and Reconstructive Surgery 110(1):177-83, July 2002.

10

Nutrition and Wound Care

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Objectives

After completing this chapter, you'll be able to:

- describe the process of nutrition screening using a validated form to identify risk for malnutrition and pressure injury
- identify the parameters involved in completing a nutrition assessment
- define the importance of nutrients for wound healing
- implement validated nutrition guidelines for the prevention and healing of pressure injuries.

Introduction

Nutrition and hydration play a key role in both the prevention and treatment of wounds. The goal in preventing pressure injury is to screen, identify, and provide appropriate interventions for individuals at risk for malnutrition and pressure injury development. A nutrition assessment should be completed both for individuals who are at high risk for developing pressure injuries and for those who currently have wounds. The data derived from the assessment should be used to develop the nutrition section of the wound treatment and/or care plan. The nutrition interventions selected to manage the current condition should be individualized, based on standard guidelines, and reviewed as new research data become available. The European Pressure Ulcer Advisory Panel (EPUAP), the National Pressure Injury Advisory Panel (NPIAP), and the Pan Pacific Pressure Injury Alliance (PPPIA) released the 2019 Pressure Injury Prevention and Treatment Clinical Practice Guidelines, which are supported by the strength of evidence for recommendation and the strength of recommendation detailed in Appendix “Definitions of the EPUAP NPIAP PPPIA 2019 Clinical Guideline Strengths of Evidence and Strengths of Recommendations.”

Impact of Undernutrition/Malnutrition

There are many risk factors for pressure injuries including malnutrition, advanced age, immobility, hyperglycemia, poor perfusion, incontinence, and multiple comorbidities.[1,2] The National Pressure Ulcer Long-Term Care cohort study of 1,524 residents in 95 nursing homes noted a higher pressure injury incidence among frail residents and those who had more severe illness, low body mass index (BMI), significant weight loss, and difficulty eating independently.[3] A retrospective study by Fry et al. of 1 million surgical patients in 1,368 hospitals in the United States showed that weight loss and/or preexisting malnutrition was a positive predictive variable in all eight major surgery–associated "never events" defined by the Centers for Medicare and Medicaid Services (CMS) including pressure injuries where the odds increased 3.8 times.[4]

Shahin and colleagues' research investigated the relationship between pressure injury and malnutrition parameters in German hospitals and nursing homes and noted a significant relationship between the presence of pressure injuries and unintended weight loss (5% to 10%) and poor nutritional intake.[5] An Australian study conducted in acute care and residential older adult facilities also confirmed that the odds ratio of having a pressure injury was higher in adults who were identified as malnourished.[6] Iizaka et al.'s study in Japan of older home care patients noted the rate of malnutrition was 58.7% for those with pressure injuries versus 32.6% for those without pressure injuries.[7] The NPIAP's 2014 Consensus Conference supported the statement that individuals with malnutrition in combination with multiple comorbidities are at increased risk for developing pressure injuries.[8]

Studies diagnosing malnutrition have used various criteria, such as anthropometric benchmarks, biochemical tests, or physical assessment parameters, to define malnutrition, often failing to differentiate between undernutrition and severe malnutrition.[9–12] Malnutrition was defined by the Agency for Healthcare Research and Quality (AHRQ) as one of the common syndromes associated with

increased risk for mortality and institutionalization that may be impacted by primary and secondary preventions.[12] Malnutrition is defined as any nutritional imbalance[13]; therefore, even overweight or obese individuals who experience a severe acute illness or major traumatic event are at risk for malnutrition. The consensus statement of the Academy of Nutrition and Dietetics (Academy) and the American Society for Parenteral and Enteral Nutrition (A.S.P.E.N.) recommends a set of diagnostic characteristics be used to identify and document adult malnutrition.[11]

Chronic starvation occurs solely as a result of inadequate nutrient intake as metabolism adapts by lowering energy expenditure, thus protecting lean body mass (LBM). Inflammation is the body's method of protecting itself when metabolic stress arises from the impact of injury, illness, or infection. Inflammation is necessary to remove damaged cells or pathogens and begin the healing process. The pathophysiology of malnutrition and the inflammatory process are associated with declining nutritional status and health. Diseases such as diabetes mellitus (DM), cardiovascular diseases, arthritis, and cancers cause chronic inflammation that is sustained, low grade, and persistent. Elevated energy expenditure and catabolism of LBM is associated with chronic inflammation. Individuals with severe acute illness or trauma experience severe inflammation and increased metabolic stress, which lead to increased energy expenditure and nitrogen excretion. This process triggered by the acute-phase inflammatory response leads to malnutrition and can limit the effectiveness of nutrition interventions.[14] The anorexia accompanying inflammation promotes continued loss of lean tissue, especially if nutrient intake is inadequate. Jensen describes the severity and length of inflammation leading to the loss of LBM and physical function as disease-related malnutrition.[14] Individuals with chronic or acute disease/injury and inflammation have elevated inflammatory markers (tumor necrosis factor, interleukin-6, and alpha interleukin-18), reduced cell mass, and loss of lean body protein.[15] A 10% loss of LBM leads to immune suppression and increases the risk of infection and at 15% to 20% will delay wound healing.[15] LBM is vital for wound healing, muscle strength, and immune and organ function. An

international guideline committee composed of members from A.S.P.E.N and the European Society for Enteral and Parenteral Nutrition (ESPEN) recommended an etiology-based approach for diagnosing adult malnutrition, which was adapted by the Academy and A.S.P.E.N.[11] Etiology-based malnutrition syndromes is based on Jensen's proposal that malnutrition is driven by various etiologies and degrees of inflammation (Fig. 10-1).

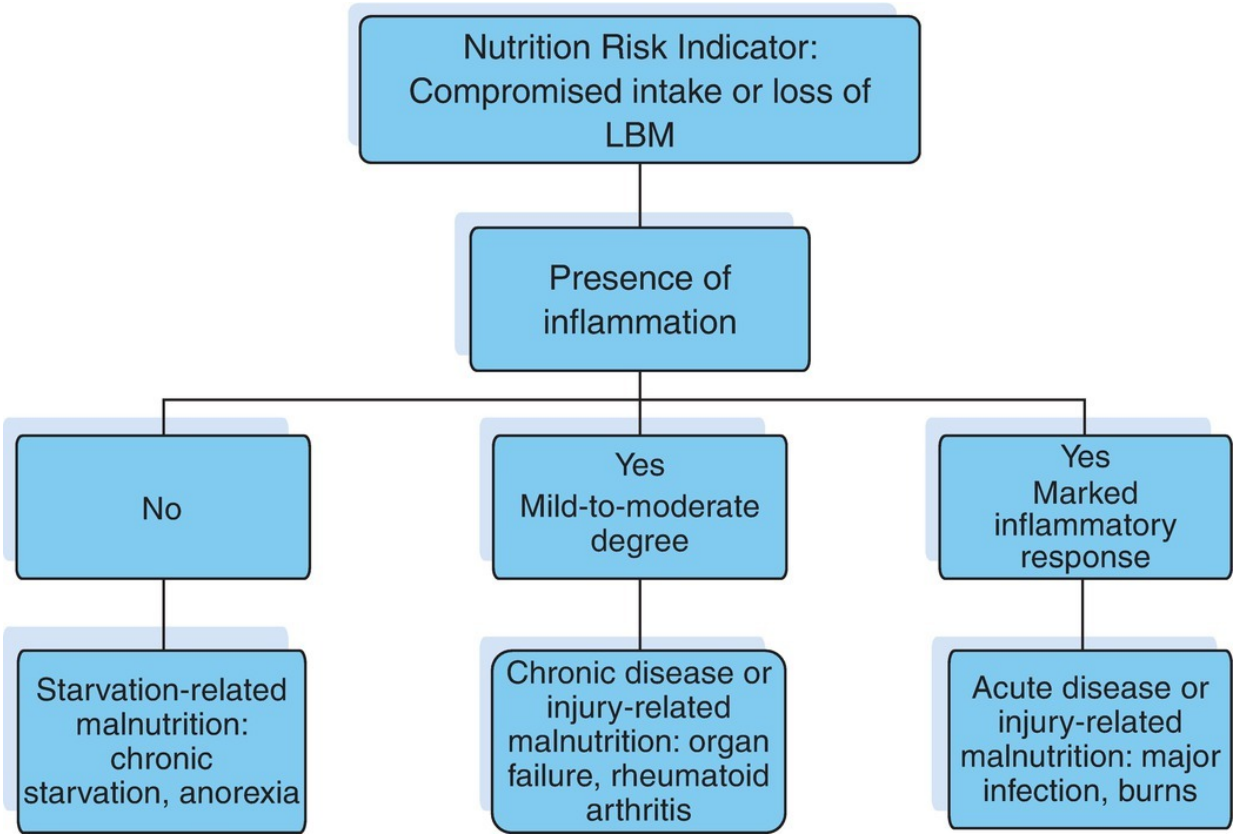

Figure 10-1. Etiology-based malnutrition.

(Adapted with permission from White, J., Guenter, P., Jensen, G., et al. "Consensus Statement of the Academy of Nutrition and Dietetics/American Society for Parenteral and Enteral Nutrition: Characteristics Recommended for the Identification and Documentation of Adult Malnutrition (Undernutrition)." *Journal of the Academy of Nutrition and Dietetics* 112:730, 2012. Copyright © 2012 American Society for Parenteral and Enteral Nutrition and the Academy of Nutrition and Dietetics. Published by Elsevier Inc. All rights reserved.)

The Academy and A.S.P.E.N consensus statement recommends identifying two or more of the following six characteristics when diagnosing malnutrition[11]:

- Insufficient energy intake based on individual's estimated needs
- Weight loss
- Loss of muscle mass
- Loss of subcutaneous fat
- Localized or generalized fluid accumulation that may sometimes mask weight loss
- Diminished functional status as measured by handgrip strength

Implementing this standardized method to diagnose the nutritional status of adults should become part of the interdisciplinary team's (IDT) protocol for prevention and/or treatment of individuals with wounds. Nutrition screening supports the concept of a standardized method to diagnose malnutrition.

ESPEN appointed a group of clinical scientists to perform a modified Delphi process with the goal of providing a consensus-based minimum set of criteria for the diagnosis of malnutrition that can be applied independent of clinical setting and etiology. The ESPEN consensus statement recommended that individuals at risk of malnutrition be identified using a validated screening tool and be assessed and treated. The result was two consensus options: option one to define malnutrition using BMI < 18.5 or option two to define malnutrition using unintended weight loss (>10% over time or >5% in 2 months) or reduced BMI or low fat-free mass.[16]

Nutrition Screening

Nutrition screening is the process of identifying characteristics that are known to be associated with nutrition problems. Its purpose is to pinpoint individuals who are malnourished or at nutrition risk and then to determine appropriate interventions based on the findings. Screening tools should be cost-effective, validated for the population served, easy for the practitioner to use, and acceptable to the individual. Any qualified member of the healthcare team, including the registered dietitian nutritionist (RDN), nutrition and dietetics technician, registered (NDTR), nurse, physician, or other qualified health professional, can complete a nutrition screening tool. The result should be documented in the medical record and communicated to the RDN and IDT. The Joint Commission requires nutrition screening to be completed within 24 hours after inpatient admission when applicable to the patient's condition.[17]

A number of validated nutrition screening tools are available, including the Mini Nutritional Assessment (MNA®), the Malnutrition Universal Screening Tool (MUST), the Malnutrition Screening Tool (MST), and the Subjective Global Assessment (SGA). The MNA® screens six areas, recent food intake, weight loss mobility, psychological stress, neuropsychological problems, and BMI, and assigns each a numerical score. The total score defines the risk of malnutrition. It has been validated in the elderly population in community and long-term care settings.[18,19] Research comparing the nutritional status of individuals with and without pressure injuries concluded that the MNA® is easy to use for assessing older adults with pressure injuries and multiple comorbidities.[20] Tsai et al. in a comparative study of six screening tools used to sample elderly people established that the MNA® had a sensitivity of 98.1% and specificity of 75% for identifying nutritional risk.[21] Grattagliano et al. followed 274 adults older than 75 years of age for 2 years to evaluate the predictive power of the MNA® on the occurrence of major events. The study confirmed that the prevalence of malnutrition is high in adults older than 75 years of age and recommended physicians

administer the MNA® and recommend interventions for improving the nutritional status of malnourished or older adults at nutrition risk.[22] The goal of this 6-month perspective observational study defining the nutrition status of patients admitted to long-term care assessed the progression of their nutrition status until discharge. Seventy-six older adults were enrolled in the study, and 15.8% had pressure injuries. After screening for nutrition status using the MNA®, 79.4% of the patients with pressure injuries were malnourished and 20.6% were at risk for malnutrition. Corrales et al.[23] concluded this study demonstrated a high prevalence of malnutrition in older adults admitted to long-term care, and its association with pressure injury incidence. The authors recommended assessing nutrition status at admission using a validated tool such as the MNA® and introducing nutrition interventions early to reduce the complications of malnutrition, such as pressure injuries.[23]

The MUST has been validated in acute care, long-term care, and community settings. Poulia et al. concluded that the MUST was the most appropriate tool for evaluating the risk of malnutrition in older adults admitted to hospitals, with a high sensitivity (87.3%) and a high negative predictive value (75%) for identifying nutritional risk.[24]

Tsaousi et al. conducted a prospective cohort study of 471 patients screened for nutrition risk using the MUST tool and noted that the prevalence of pressure injuries was 14.5%. Patients with pressure injuries were older, had a lower BMI, and had unintended weight loss and poor appetites. They had longer hospital stays, thus increasing the cost of care. The authors concluded that recognizing malnutrition quickly and implementing nutrition care was important for patients at risk for or with a pressure injury.[25]

The Nutrition Risk Screening (NRS) 2002 is also a validated screening tool for adults in hospitals.[26] The Short Nutritional Assessment Questionnaire (SNAQ) has been validated in both the hospital and residential care. Weight change, appetite, supplements, and tube feeding are the parameters of the SNAQ.[27]

When the screening tool triggers risk for or actual malnutrition, undernutrition, or unintended weight loss, a timely referral to the RDN

is critical for nutrition assessment and intervention.

Conditions that require immediate nutrition assessment and intervention include the following:

- Unintended weight loss: 5% in 30 days, or 10% in 180 days
- Disease states and conditions: diabetes, malabsorption, dementia, chronic obstructive pulmonary disease (COPD), cancer, or renal disease
- Immobility and inactivity: hip fracture, spinal cord injury, amyotrophic lateral sclerosis (ALS), and stroke
- Chewing and swallowing difficulties (dysphagia) resulting from stroke, Parkinson's disease, cerebral palsy, or other conditions

Altered mental status often limits an individual's ability to eat independently or to comprehend the importance of consuming a balanced diet. Advanced dementia can result in weight loss, dysphagia, and malnutrition. When individuals become incapable of responding to caregivers' assistance to nourish them, this can lead to unintended weight loss, which may in turn increase their risk for pressure injury development.

Immobility affects an individual's ability to purchase food, prepare meals, or travel to a restaurant or congregate setting for meals and sometimes affects ability to enjoy food or metabolize food properly. This may result in the consumption of a diet lacking in the proper nutrients. For example, hip fracture and spinal cord injury restrict mobility, and hip fracture often results in increased pain that makes it difficult for the individual to concentrate on consuming healthy meals.

Nutrition Assessment

Nutrition assessment is a systematic process of obtaining, verifying, and interpreting data in order to make decisions about the nature and cause of nutrition-related problems. It is an ongoing process that involves initial data collection followed by continued reassessment and analysis of the individual's status compared with specific criteria.[28] The (Academy's) Nutrition Care Process (NCP), which is designed to improve the quality and consistency of individualized nutrition care for individuals, should be followed by RDN and NDTR in all healthcare settings.[28] The NCP consists of four steps: nutrition assessment, nutrition diagnosis, nutrition intervention, and nutrition evaluation and monitoring. RDNs and NDTRs are the only healthcare professionals who utilize the NCP. The nutrition diagnosis, not a medical diagnosis, involves diagnosing and classifying a nutrition problem that the RDN is responsible for treating independently. An example of a nutrition diagnosis is the following: Unintentional weight loss related to oral intake not meeting energy needs as evidenced by weight loss of 5% in 3 weeks. The nutrition intervention step is meant to solve a nutrition diagnosis or problem. The RDN discusses the proposed intervention with the IDT including the individual and/or family/caregiver. An example of a nutrition intervention is as follows: Provide six small meals per day plus high-calorie supplement at bedtime, weigh weekly, and monitor intake and weights. The nutrition monitoring step notes the progress of the nutrition intervention to determine if the expected outcome is progressing or achieved. Nutrition interventions should be individualized and adjusted if the desired outcome is not achieved.

The NCP identifies five domains for nutrition assessment: Food/Nutrition Related History, Biochemical Data, Anthropometric Measurements, Nutrition-Focused Physical Findings, and Client History. Nutrition assessment also includes interpretation of data from the screening process as well as a review of entries from other disciplines (such as speech, occupational, or physical therapy) that may affect the evaluation process. Nutrition assessment leads to

identification of a nutrition diagnosis or problem that can be resolved or improved through treatment (nutrition intervention).

Food/Nutrition Related History

The RDN should evaluate the adequacy of the individual's current and previous food intake and meal pattern including any supplements, food intolerances, allergies, and cultural food preferences. Individuals receiving treatment at a wound care center may need to complete a 24-hour recall based on verbal self-reporting of everything they ate or drank during a specified time. While this method may not be accurate, the RDN could gather information about their eating habits. Another method to measure intake is a food diary record, which is a self-administered, subjective dietary intake questionnaire that collects detailed information about all types of food and beverage consumed over a specified period of time. This type of tool does not rely on memory but may require a higher level of literacy.

As part of the nutrition assessment, observation during mealtime gives the healthcare professional the opportunity to determine whether the individual has self-feeding, chewing, or swallowing problems that may require either speech or occupational therapy screening and/or treatment. Inability to feed oneself has been identified as a risk for unintended weight loss.[29] Adequate consumption of food and fluid is also a concern for those with swallowing problems, which places individuals at risk for malnutrition and pressure injuries. The speech language pathologist (SLP) screens, assesses, and defines the food texture and fluid consistency as well as the need for individualized feeding techniques such as positioning for ease of swallowing. These interventions are implemented by the dining services department and nursing staff and monitored and evaluated by the RDN. For example, individuals may require thickened liquids to prevent aspiration. The occupational therapist determines the appropriate self-help feeding devices to promote eating independence. Similarly, physical therapy sessions often result in the need for both increased calories and fluid, which the RDN will calculate and arrange to provide in the form of food and/or supplements at appropriate times.

Medications can influence an individual's nutrition status and have been identified as a cause of weight loss.[30] Drugs may either inhibit or

induce metabolism of a nutrient or increase the excretion of a nutrient. Medicine designed to calm and reduce agitation may in turn reduce mobility and activity levels and place individuals at risk for pressure injury development. Medications may increase or decrease appetite, alter sense of taste or smell, or cause gastric disturbances. Drug therapy may trigger adverse effects; for example, antibiotics often cause nausea and gastric disturbances that curtail an individual's food and fluid intake. The list of medications and herbal supplements that may influence nutrient intake or interfere with nutrition is extensive. Consult a validated reference to identify potential food–medication interactions or nutrition concerns associated with medication and herbal supplements.[31]

Biochemical Data

Biochemical tests are evaluated as part of the nutrition assessment process. However, there is no one test specific to nutrition status.

Albumin is a serum protein with a large body pool size distributed between the vascular and interstitial spaces, with 50% located in extravascular spaces. It functions as a carrier protein and assists in maintaining oncotic pressure. Only 5% of albumin is synthesized every day by the liver; therefore, protein intake does not affect the albumin pool on a daily basis. The majority of the changes in albumin are caused by the redistribution between the intravascular and extravascular spaces. For example, edema, ascites, and inflammation depress albumin levels, and dehydration falsely elevates albumin.

Prealbumin (transthyretin) is a transport protein for thyroxine and a carrier for retinal binding protein. Prealbumin levels are elevated in acute renal failure as the kidneys degrade it. It is also affected by some of the same factors as albumin.

Transferrin has been touted as a marker of nutritional status, but since it is involved in iron transport, its levels are affected by iron status.

Albumin, prealbumin, and transferrin are negative acute-phase reactants that are stimulated by the inflammatory process including infection, trauma, surgery, autoimmune processes, burns, etc. Acute-phase proteins change by approximately 25% during inflammation and are expected to return to normal once the inflammatory response resolves. The response in acute and chronic inflammation is a release of cytokines such as interleukin-6 that is responsible for the production of most acute-phase proteins.[32] Cytokines are responsible for fever, inflammation of chronic disease, and cachexia. However, serum albumin levels may drop in as few as 8 hours in severe stress or inflammatory conditions even when protein intake is adequate. The Academy's Evidence Analysis Library analyzed changes in albumin and prealbumin for several conditions and indicated that these acute-phase proteins do not consistently or predictably change with weight loss, calorie restriction, or nitrogen balance. They reflect severity of

the inflammatory response rather than nutritional status.[33] For this reason, a decrease in serum albumin is increasingly seen as a poor reflection of nutritional status.[11,32–38] Low serum protein levels may indicate that the individual is ill and therefore at risk for malnutrition. Frequent monitoring of weight status and oral intake would be important, especially if the individual is at risk for impaired skin integrity.

Other biochemical data that should be evaluated include glucose levels (fasting blood sugar and A1c) for persons with diabetes along with renal function to determine if individuals can tolerate the increased protein that is recommended for wound healing.

Anemia, is defined as a blood disorder in which there is a reduced size or number of red blood cells or low hemoglobin, and low hematocrit, which can have a negative effect on wound healing. When anemia is present, the blood has reduced oxygen-carrying capacity leading to various side effects and negative consequences such as lower endurance, poor temperature regulation, and decreased immune function. Individuals with infections or other inflammatory issues may have low hemoglobin and hematocrit levels associated with a redistribution of iron rather than a change in nutrition intake.[39] There are different classifications of anemia, and the type should be identified in order to provide the appropriate nutritional intervention.

The nutrition assessment should focus on identifying risk factors that may contribute to the development of dehydration (Table 10-1).[40–42] Dehydration can have serious consequences especially for the frail older adult: decreased functional ability, predisposition to falls, infections, and fluid and electrolyte imbalances. The dehydrated individual exhibits unintended weight loss (2%, mild; 5%, moderate; and 8%, severe), dry skin and mucous membranes, rapid pulse, decreased venous pressure, subnormal body temperature, low blood pressure, and altered sensation.

Table 10-1 Signs of Dehydration

Sufficient hydration is essential for all individuals and even more so for the individual with a wound. Use the following guidelines to prevent dehydration—and to recognize and treat it should it occur.

Preventing Dehydration

- If the patient can drink independently, keep water or other beverages at bedside so that they are easily accessible and in a container the individual can handle easily.
- If the individual doesn't consume fluids on his or her own, offer water at least every 2 h.

Symptoms of Dehydration

- Mild dehydration: increased heartbeat, increased respirations, decreased sweating, urination, and increased body temperature, extreme fatigue, nausea, and tingling of the limbs
- Severe dehydration: muscle spasms, confusion, seizures, and other symptoms

Note: Because some older adults have confusion related to other diagnoses, it is important to look for all symptoms of dehydration, not just disorientation.

Monitor dehydration by looking for:

- dry skin, dry tongue, cracked lips, dry mucous membranes
- complaining of thirst (often diminished in elderly individuals)
- poor fluid intake, recent decline in oral intake
- poor skin turgor (Note: the pinch test for skin turgor may be an unreliable indicator of dehydration in elderly individuals. If you use this test, use only the skin on the forehead or sternum, and pinch gently. If well hydrated, the skin goes back into place in 2 s.)
- fever
- appetite loss
- dizziness upon sitting or standing
- confusion or change in mental status
- laboratory values (see Table 10-2)
- decrease in blood pressure
- increase in pulse rate
- newly present constipation, fecal impaction (Note: recent diarrhea may explain the dehydrated state, and constipation is common when dehydration exists.)
- recent decrease in urine volume or more concentrated urine than normal
- lethargy
- functional decline
- fluid and electrolyte imbalance
- significant weight loss in a short period of time[40–42]

Individuals who are at risk of dehydration require careful monitoring, such as daily weights. A weight loss of 2 kg in 48 hours indicates a corresponding loss of 2 L of fluid. Elderly patients, whose sense of thirst often declines, should be offered fluids more frequently. The American Medical Directors Association published a guideline, which may be helpful to those working with older adults for the management of dehydration.[43] Laboratory results that may be indicators of dehydration include elevated albumin, BUN, hemoglobin and/or hematocrit, BUN/creatinine ratio greater than 25.1 and increased serum osmolality, serum sodium, urine concentration, and/or urine specific gravity.[44]

Anthropometric Factors

Anthropometry, the measurement of body size, weight, and proportions, is used to evaluate an individual's nutritional status. A change in anthropometric values can signal problems such as wasting or edema, reflecting nutritional excess or deficit. Accurate heights and weights are critical as they are the basis for determining caloric and nutrient requirements. Adjustment or notations should be made for casts and other appliances that alter true weight.

Practice Point

Patients should be weighed each time on the same scale, at the same time of day, and with minimal clothing. Care should be taken to subtract wheelchair weights, if weighing on a wheelchair scale, noting the weight of any casts or braces (and subtracting these weights if they cannot be removed before taking the weight).

BMI is a weight-to-height ratio derived from body weight in kilograms divided by the square of the height in meters:

$$\frac{\text{Weight (kg)}}{\text{Height (m}^2\text{)}}$$

or

$$\text{BMI} = \frac{\text{Weight (pounds)}}{\text{Height (inch}^2\text{)}} \times 705$$

A normally hydrated person with a BMI > 30 is considered obese (see Chapter 24 Adiposity and the Bariatric Patient). The National Heart, Lung, and Blood Institute considers a BMI < 18.5 underweight for adults, which may place the individual at nutrition risk.[45]

One measurement of determining the severity of undernutrition/malnutrition is any deviation from usual body weight.

$$\frac{\text{Usual weight} - \text{current weight}}{\text{Usual weight}} \times 100 = \%\,\text{Weight Loss}$$

For example, a usual body weight of 145 pounds that decreased to 137 pounds in a 30-day period is a 5.5% loss.

When evaluating the severity of weight variances, it is important to determine whether the weight loss was intended or unintended. If the weight loss was unintended, it is essential to determine possible causes, such as recent surgery, diuretic therapy, or other new treatments, that may affect weight status. Unintended weight loss can signify malnutrition and increase the risk of mortality. Weight loss, especially in older adults, results in undernutrition and increases the risk of mortality. A 5% loss in 30 days or a 10% decline in 6 months increases the risk of adverse outcome including pressure injuries.[25]

Nutrition-Focused Physical Assessment

The nutrition-focused physical assessment completed by the RDN examines the individual for signs of malnutrition. The RDN begins with a general observation of the individual and focuses extensively on body composition such as muscle and skin condition, checking for loss of subcutaneous fat as evidenced by loose skin in the extremities and observing for listlessness, muscle wasting, the presence of peripheral edema in the absence of cardiac disease or circulatory disorder, and dull, dry, sparse hair that can signify a possible protein–energy deficiency.

The oral cavity is examined for missing or decayed teeth, swollen gums that may result in difficulty chewing or swallowing, affecting the individual's ability to ingest adequate calories and fluids. Sensory problems are also evaluated because declining hearing and vision can compromise a person's communication skills, often resulting in reduced caloric intake.

The older adult is particularly prone to pressure injuries as a result of decreased mobility, multiple comorbid conditions, poor nutrition, and loss of muscle mass. In addition to malnutrition and unintended weight loss, nutritional factors thought to contribute to skin breakdown include protein deficiency, which creates a negative nitrogen balance; anemia, which inhibits the formation of red blood cells; and dehydration, which causes dry, fragile skin.

Immune function declines with age, thus increasing the risk of infection. With advancing age also comes decreased skin response to temperature, pain, and pressure. This affects the skin's elasticity and the healing process.

The nutrition assessment information collected is documented in the medical record, and an individualized interdisciplinary care plan is established.

Table 10-2 outlines nutrition screening, assessing, and care planning recommendations from the 2019 of EPUAP, NPIAP, and

PPPIA, Prevention and Treatment of Pressure Ulcers/Injuries Clinical Practice Guidelines.[46]

Table 10-2 Nutrition Screening, Assessment, Care Planning EPUAP NPIAP PPPIA 2019 Pressure Injury Practice Guideline Recommendations

Recommendations: Strength of Evidence (SOE) and Strength of Recommendation
4.1 Conduct nutritional screening for individuals at risk for pressure ulcer/injury. **SOE = B1; Strength of Recommendation = ↑↑**
4.2 Conduct a comprehensive nutrition assessment for adults at risk of a pressure ulcer/injury who are screened to be at risk of malnutrition and for all adults with a pressure ulcer/injury. **SOE = B2; Strength of Recommendation = ↑↑**
4.3 Develop and implement an individualized nutrition care plan for individuals with, or at risk of, a pressure ulcer/injury who are malnourished or who are at risk of malnutrition. **SOE = B2; Strength of Recommendation = ↑↑**

Adapted with permission from European Pressure Ulcer Advisory Panel, National Pressure Injury Advisory Panel and Pan Pacific Pressure Injury Alliance. In: E. Haesler, ed. *Prevention and Treatment of Pressure Ulcers/Injuries: Clinical Practice Guideline. The International Guideline*. EPUAP/NPIAP/PPPIA, 2019.
See Appendix “Definitions of the EPUAP NPIAP PPPIA 2019 Clinical Guideline Strengths of Evidence and Strengths of Recommendations.”

Role of Nutrition in the Cascade of Wound Healing

The four phases of wound healing are a cascade of overlapping events that include hemostasis, inflammation, proliferation, and maturation. Hemostasis occurs with the disruption of tissue following an injury and stops hemorrhage at the site of blood vessel damage. Inflammation begins following an acute injury and includes the characteristic signs of tissue damage of pain, heat, and swelling. The proliferative stage begins 3 or 4 days after injury and can last for 2 or 3 weeks. Epithelization, granulation, and angiogenesis occur in the proliferative stage. During the maturation or rebuilding stage, which can last for a year or longer, cells contract and form an adhesive mechanism to bind the collagen network and bring the wound edges closer together.[47] Refer to Chapter 5 for the complex details of the wound healing process. All phases of wound healing require protein, which is considered the building block for tissue repair and healing. Table 10-3 describes the nutrients involved in the phases of wound healing.[47]

Table 10-3 Phases of Wound Healing: Nutrients and Their Function

Phases of Wound Healing	Nutrients and Function
Hemostasis	**Protein:** required for all stages of wound healing **Vitamin C:** helps maintain hemostasis and promotes inflammatory process
Inflammation	**Protein:** cell proliferation and production of granulation tissue **Vitamin C:** antioxidant and helps wound transform from inflammatory to proliferative stage **Vitamin K:** cofactor for enzymes involved in clotting factors **Zinc:** inadequate supply can delay healing **Copper:** lymphocyte synthesis can be reduced with copper deficiency caused by excess zinc intake **Vitamin A:** necessary for synthesis of immune cells
Proliferation	**Protein:** collagen deposition, supplementation associated with improved healing **Amino acids:** arginine and glutamine become conditionally essential during proliferation **Vitamin A:** facilitates collagen synthesis and promotes epithelization **Vitamin C:** collagen formation **Vitamin D:** diverse effects including cellular differentiation and proliferation; note: lower levels noted in venous and diabetic foot ulcers and increased levels reduced size of wounds in 1 study **Iron:** hemoglobin synthesis
Maturation	Adequate energy, protein and micronutrients as noted above and per research-based guidelines are required

Wound Healing. The ASPEN Adult Nutrition Support Core Curriculum, Chapter 12, 3rd ed. 2017. Available at: www.amazon.com/ASPEN-Adult-Nutrition-Support-Curriculum/dp/1889622311. *Accessed March 3,* 2020.

Function of Nutrients

There are six major classes of nutrients: carbohydrates, proteins, fats, vitamins, minerals, and water. Through the process of metabolism, organic nutrients are broken down to yield energy, rearranged to build body structures, or used in chemical reactions for body processes.

Individuals at risk of malnutrition and pressure injuries or who currently have wounds require additional calories to compensate for the increased metabolic stress due to hypermetabolism. Stress as a result of injury, surgery, burn, fracture, or wounds results in depletion of the nutrient stores required for healing. The current caloric recommendation is 30 to 35 calories/kg body weight for adults with a pressure injury who are malnourished or at risk of malnutrition.[46]

Carbohydrate

Carbohydrates provide energy and prevent gluconeogenesis, which occurs when the body is forced to convert protein stores for energy. Approximately 50% to 60% of an individual's total caloric intake should come from carbohydrates. Glucose is the main source of fuel for collagen synthesis required for wound healing. Carbohydrate sources should be primarily from complex sources such as whole grains, fruits, and vegetables, which also provide important nutrients including dietary fiber, vitamins, and minerals.

Protein and Amino Acids

Protein accounts for 17% of an individual's body weight, mainly in the form of lean tissue, muscle, and connective tissue. Half of the body's protein consists of collagen, actin, and myosin, which provide structure to the body, and hemoglobin, which transports oxygen to the cells. Protein is responsible for repair and synthesis of enzymes involved in wound healing, cell multiplication, and collagen and connective tissue synthesis. It is essential for promoting positive nitrogen balance, and increased protein levels have been linked to improved healing rates.[48,49] The recommended dietary allowance (RDA) for all adults, including older adults, is 0.8 g/kg body weight. Evidence supports the concept that LBM can be better maintained when an older adult consumes higher levels of protein in the range of 1.0 to 1.2 g/kg/d.[50,51] A report by the PROT-AGE Study Group suggested that individuals with severe injury or illness might need 2.0 g/kg/body of protein daily.[52] The 2019 EPUAP/NPIAP/PPPIA guideline recommendation is 1.25 to 1.5 g protein/kg body weight for adults with a pressure injury, who are malnourished or at risk of malnutrition.[46] Clinical judgment should be used to determine individual protein requirements, considering the number and severity of wounds, renal function, and comorbidities, as well as tolerance to interventions. For example, high protein levels may not be appropriate for individuals with chronic kidney disease (CKD). Lean meats, poultry, fish, low-fat dairy products are healthy sources of dietary protein. Oral nutritional supplements (ONSs) provide good alternatives for protein sources when dietary intake is inadequate.

When the body is under stress, glutamine, cysteine, and arginine become conditionally indispensable amino acids. L-arginine is composed of 32% nitrogen, and its function is to stimulate the insulinlike growth factor that is involved in wound healing. Arginine enhances immune function and wound collagen deposition in healthy elderly people.[53] A 12-week RCT study by Cereda et al. in four nursing homes concluded that the rate of pressure injury healing accelerated when a nutritional formula enriched with protein, arginine, zinc, and antioxidants was consumed daily for at least 8 weeks

compared to an isocaloric–isonutriogenous formula.[54] An economic evaluation was conducted alongside the multicenter study. While the disease-specific formula was more expensive, the increased healing rate with the arginine-enriched formula was cost-effective, reducing direct nursing care, treatments, and dressings.[55] Another RCT by van Anholt et al.[56] investigated the same formula used in the Cereda study[55] to improve healing in well-nourished older adults with stage 3 and 4 pressure injuries. Participants in the trial were from eight healthcare centers, hospitals, or nursing homes in four European countries. The study concluded that a high-protein supplement with arginine, zinc, and micronutrients was associated with improved healing rate in older adults who were not malnourished.[56]

After 3 weeks of consuming a high-calorie ONS containing arginine, zinc, and vitamin C, individuals in one study had a significant reduction in the Pressure Ulcer Scale for Healing (PUSH) score compared to no improvement in the PUSH score for those consuming either a standard diet or an ONS without arginine and micronutrients.[57]

Fats and Fatty Acids

Fat is the most concentrated source of energy and provides a reserve source of energy in the form of stored triglycerides in adipose tissue. Fat calories should constitute 20% to 25% of total caloric intake. Vegetable, olive, and nut oils as well as nuts, nut butters, seeds, and fatty fish provide healthy sources of polyunsaturated and monounsaturated fat.

Fat-Soluble Vitamins

Fat-soluble vitamins A, D, E, and K remain in the liver and fat tissue of the body until used. Because the body does not excrete excess fat-soluble vitamins, the risk of toxicity from overdose exists.

Practice Point

Older adults may have a difficult time consuming adequate amounts of calcium, vitamin D, and vitamin B_{12} without supplementation. Individuals living alone or on a fixed income may not consume a balanced diet. If a vitamin or mineral deficiency is confirmed or the diet intake is extremely inadequate, a multivitamin may be warranted.

Vitamin A is responsible for epithelium maintenance. During the inflammatory phase, it increases the number of macrophages and monocytes in the wound and stimulates cellular differentiation in fibroblasts and collagen formation.[58] Vitamin A deficiency, which is uncommon, may result in delayed wound healing and increased susceptibility to infection. The wound healing process may be compromised for individuals taking steroids; however, there is limited research to support mega doses of vitamin A to accelerate healing.

Vitamin E is an antioxidant and is responsible for normal fat metabolism and collagen synthesis and does not appear to play an active role in wound healing.[59]

Water-Soluble Vitamins

Water-soluble vitamins C and B play a role in wound healing. Vitamin C is an antioxidant essential for collagen synthesis, aids in iron absorption and activation of copper, and plays an important role in immune function and protein metabolism.[60] Collagen and fibroblasts compose the basis for the structure of a new wound bed, so a vitamin C deficiency delays healing. A deficiency of vitamin C prolongs healing time and contributes to reduced resistance to infection.[60] However, there is no clinical evidence that wound healing is improved by providing doses of vitamin C above the dietary reference intake (DRI) of 70 to 90 mg/d. A multicenter, blinded trial of 88 patients with pressure injuries who were randomized to either 10 or 500 mg of vitamin C twice daily failed to demonstrate any improved healing or closure rate between groups.[61]

Coenzymes (B vitamins) are necessary for the production of energy from glucose, amino acids, and fat. Pyridoxine (vitamin B_6) is important for maintaining cellular immunity and forming red blood cells. Thiamine and riboflavin are needed for adequate cross-linking and collagenation, but their effect has not been demonstrated in pressure injuries.

Minerals

Minerals also contribute to a patient's well-being. Zinc, a cofactor for collagen formation, also metabolizes protein, liberates vitamin A from storage in the liver, interacts with platelets in blood clotting, and assists in immune function.[62] Deficiency may occur rapidly through wound drainage or excessive gastrointestinal fluid loss or from long-term poor dietary intake. Albumin transports zinc through the body, so zinc absorption declines as plasma albumin declines (e.g., with infection, sepsis, or trauma). No clinical evidence exists to support supplementation (such as with zinc sulfate 200 to 300 mg daily, which contains more than 50 mg of elemental zinc). In a small study of individuals with pressure injuries, no effect on injury healing was seen at 12 weeks in zinc-supplemented versus non–zinc-supplemented individuals.[63] The daily required intake (DRI) for zinc is 8 to 11 mg, and the maximum daily intake or tolerable upper intake level for elemental zinc is 40 mg.[64] High serum zinc levels may inhibit healing, impair phagocytosis, and interfere with copper metabolism.[65]

Copper is essential for preserving the strength of the skin, blood vessels, and epithelial and connective tissue throughout the body. The ratio of copper to zinc is clinically more important than the concentration of either of these trace minerals.

Iron is needed for hemoglobin, collagen formation, and oxygen transport. Iron is essential for new cell generation, amino acids, and hormones. Individuals who consume a diet low in nutrient-rich foods, those who are food insecure, or individuals with poor nutrient absorption or metabolism may not be consuming an adequate diet to meet established nutritional reference standards.

A multivitamin with 100% of the DRI for minerals is the general recommendation if the diet is inadequate or if deficiencies are suspected or confirmed. Many of the oral supplements, enteral formulas, and fortified foods recommended for individuals with wounds contain additional micronutrients that should be considered before recommending additional supplementation.

Water

Water constitutes about 60% of an adult's body weight. It is distributed in the body in three fluid compartments (intracellular, interstitial, and intravascular). Water serves many vital functions in the body, including:

- aiding in hydration of wound sites and in oxygen perfusion,
- acting as a solvent for minerals, vitamins, amino acids, glucose, and other small molecules and enabling them to diffuse into and out of cells, and
- transporting vital materials to cells and removing waste from cells.

Individuals with draining wounds, emesis, diarrhea, elevated temperature, or increased perspiration need additional fluids to replace lost fluid. Individuals on air-fluidized beds may require additional fluids daily. Total fluid needs are met from the water content of food plus liquids. Food accounts for 19% to 27% of the total fluid intake of healthy adults (see Tables 10-4 and 10-5).[70]

Table 10-4 Determining Fluid Needs

There are a number of mathematical equations commonly used in practice to estimate fluid needs including:

- 1 mL/kcal of estimated needs OR 1 mL/kcal of intake
- 1,500 mL for the first 20 kg +15 mL/kg for each additional kg >20 kg
- Weight-based estimates
  - 25 mL/kg for heart failure or renal disease
  - 30 mL/kg for average adults
  - 35 mL/kg for patients with infection or draining wounds

Factors that may increase fluid needs include burns, certain medications (i.e., diuretics), circulating air bed, dehydration, diarrhea, emesis, fever, extraordinary gastric and/or renal losses, hyperventilation, hyperthyroidism, moderate or profuse perspiration, polyuria, pressure injuries or other draining wounds, or fistulas.	**Factors that may decrease fluid needs** include edema, fluid overload, heart failure, hepatic failure with ascites, severe renal failure, SIADH, and third spacing of fluids.[66–69]

Table 10-5 Nutrients and Their Therapeutic Properties

Nutrient	Function	Source
Calories	Supply energy, prevent weight loss, preserve lean body mass.	Carbohydrate, protein, and fat, with carbohydrate and fat the preferred sources
Carbohydrates	Deliver energy. Energy needs must be met to spare protein from being used for energy. Glucose supports cell growth, fibroblasts, and leukocytes.	Grains, fruits, and vegetables, with complex carbohydrate the preferred source
Protein	Contains nitrogen, which is essential for wound healing. Is a component of the immune system. Supplies the binding material of skin, cartilage, and muscle.	Meats, fish, poultry, eggs, legumes, milk, and dairy products; favor lean meat and reduced-fat or low-fat dairy products
Fat	Most concentrated energy source, carries the fat-soluble vitamins, provides insulation under the skin and padding of bony prominences, helps modulate inflammation and the immune response.	Meats, eggs, dairy products, and vegetable oils
Fluids	Solvent for minerals and vitamins, amino acids and glucose; help maintain body temperature; transport materials to cells and waste products from cells; maintain skin integrity.	Water, juices, beverages; fruits and vegetables contain ~95% water Most supplements are 75% water
Vitamin A	Important for protein synthesis, collagen formation, maintenance of epithelium, immune function. May also delay healing in older adults on corticosteroids. UL is 3,000 μg; DRI females older than 70 y is 700 μg, males older than 70 y is 900 μg.	Beef liver, milk, dark green and yellow vegetables, especially carrots, sweet potatoes, broccoli, spinach, apricots
Vitamin C	Water-soluble, noncaloric organic nutrient essential for collagen formation; enhances activation of leukocytes and macrophages to the wound site; improves tensile strength; aids in absorption of iron.	Citrus fruits and juices, tomatoes, potatoes, broccoli
Vitamin E	Antioxidant important in metabolism of fat, collagen synthesis, stabilization of cell membranes.	Vegetable oils, sweet potatoes
Copper	Inorganic, noncaloric nutrient; assists in the formation of red blood cells; responsible for collagen cross-linking and erythropoiesis. UL is 10,000 μg. DRI females and males 70 y and older is 900 μg.	Nuts, dried fruit, organ meats, dried beans, whole grain cereal
Iron	Transports oxygen to the cells as a component of hemoglobin, important in collagen formation, creates energy from cells.	Heme iron: meats, poultry, and fish Nonheme iron: vegetables, grains, eggs, meat, fish
Zinc	Inorganic, noncaloric nutrient; cofactor for collagen formation; metabolizes protein; assists in immune function; liberates vitamin A from the liver; interacts with platelets in blood clotting. Mega doses of zinc may inhibit healing and cause copper deficiency. UL is 40 mg. DRI for females 70 y and older is 8 mg, males 70 y and older is 11 mg.	Meats, liver, eggs, and seafood

DRI, Dietary Reference Intake; UL, Tolerable Upper Intake Level.

Used with permission Bernstein, M., Munoz, N. *Nutrition for the Older Adult*, 2nd ed. Burlington, MA: Jones & Barlett Learning, 2019.

Nutrition Requirements for Different Types of Wounds

The characteristics and physiology of surgical wounds, pressure injuries, and chronic wounds differ.[66,67] It is important to understand that the keys to wound healing are not the same for a chronic wound versus a wound caused by trauma. Trauma patients tend to be younger, healthier, and well nourished, whereas patients with chronic wounds tend to be older, with multiple comorbidities, and often chronically malnourished and fragile.[68,69] Therefore, medical nutrition therapy of an acute wound can be different than that of a chronic wound. However, the fundamentals of managing healing for one type of wound may be applicable to management of other wounds.[71,72]

For surgical wounds, Huckleberry suggests caloric needs between 25–30 kcal/kg/d and 1.2–2.0 g/kg/d postoperative protein requirements.[73] For individuals who have an existing pressure injury, the EPUAP/NPIAP/PPIA currently recommends calories of 30 to 35 kcal/kg/d and protein of 1.25 to 1.5 g/kg/d.[74] Individuals with a chronic wound have caloric needs of 30 to 40 kcal/kg/d and protein needs of 1 to 2 g/kg/d for individuals with chronic wound, according to Molnar (see Table 10-6).[71,72]

Table 10-6 Wound Type/Caloric/Protein Recommendations

Types of Wounds	Daily Caloric Needs	Daily Protein Needs
Surgical	25–30 kcal/kg	1.2–2 g/kg
Pressure Injury	30–35 kcal/kg	1.25–1.5 g/kg
Chronic	30–40 kcal/kg	1–2 g/kg[46,47,66–68]

Diabetes

Individuals with diabetes frequently have a higher rate of complications, including infections, which can both cause and affect poor wound healing.[75] Hyperglycemia may impair leukocyte function, thus lengthening the inflammatory process and the resolution of infection. For individuals with diabetes, a glycosylated hemoglobin (HbA1c or A1c) test is the best indicator of glucose status, as it indicates blood glucose control over the previous 3 months. Marston noted that failure to manage uncontrolled HbA1c levels impedes healing of diabetic foot injuries.[76] Effective glycemic control requires an interdisciplinary approach that addresses key areas including dietary management with educations provided by the RDN and/or certified diabetes educator.

In addition, Smart, Al Ghareeb, and Smart conducted a cross-sectional study to compare 25-hydroxyvitamin D (25[OH]D) and glycemic control with quantifiable wound healing parameters.[77] They found that subjects with both poorly controlled diabetes and vitamin D deficiency were more likely to exhibit delayed wound healing or present with deteriorating wound. Furthermore, delayed diabetic food injury healing in this study was linked with vitamin D deficiency, poor glycemic management, and exposed bone in the wound bed in this study. The authors noted that an RCT study as a follow-up to this cross-sectional study could validate the causal link supported by their research.[77]

The primary nutrition goal for individuals with diabetes is to improve metabolic control of glucose and lipids and to prevent or delay complications of diabetes. The American Diabetes Association (ADA) notes there is no “one size fits all” eating pattern for individual with diabetes. A variety of eating patterns have been shown to have a modest effect in managing diabetes including the Mediterranean-style diet, Dietary Approaches to Stop Hypertension (DASH) eating plan, plant-based vegan or vegetarian eating patterns, and lower-fat and lower-carbohydrate diets. Consistent carbohydrate intake with respect to the time and amount of insulin can result in improved glycemic control and reduce the risk of hyperglycemia, and this has an

evidence rating of B in the ADA's medical nutrition therapy guidelines.[78] An individual with diabetes who also has a pressure injury should receive a consult to an RDN for a nutrition assessment and nutrition plan based on his or her individual goals and desired outcomes.

Chronic Kidney Disease

Individuals with CKD often have multiple medical conditions, such as diabetes or heart disease, which complicates the nutrient parameters of their diet. For example, a CKD diet that is limited in calories, protein, potassium, phosphorus, sodium, and fluid often results in poor dietary intake that may not meet the nutrient requirement for an individual with wounds. The Academy's position paper on individualized nutritional approaches for older adults in long-term care, postacute care, and other settings advocates for RDNs to assess and evaluate the need for nutrition intervention tailored to each person's medical condition, needs, desires, and rights.[79]

Obesity

Obesity levels have risen dramatically in the last few decades and in 2014, more than 1.9 billion adults worldwide were overweight and 600 million were obese.[80] The World Health Organization (WHO) defines overweight and obesity as abnormal or excessive fat accumulation that may impair health and notes that obesity is associated with greater morbidity than being underweight.[81]

Obesity is a complex, multifactorial, and largely preventable disease, which increases the risk of impaired cutaneous wound healing, fascial dehiscence, surgical site infections, and vascular disease.[80] In obesity, capillary density fails to meet the demand of adipose tissue because adipose tissue disproportionately outgrows capillary density. In addition, capillary proliferation is restricted due to increased levels of fibrosis. Larger blood vessels in obese individuals that do not deliver oxygen as efficiently as do capillary blood vessels, decrease tissue perfusion and oxygenation because of the greater diffusion barrier.[80]

Metabolic demand is increased in obese individuals. The heart needs to sufficiently perfuse peripheral tissues in order to prevent ischemia. Patients who are Class 3 or extremely obese (BMI ≥ 50 kg/m^2) have increased risk of perioperative complications.[82] These individuals should be screened for diabetes and provided with medical nutrition therapy when indicated. However, in 2016, the American Society for Metabolic and Bariatric Surgery no longer support presurgical weight loss as an insurance requirement because of lack of evidence on postoperative outcome.[82]

Nutrition screening and assessment are important to determine if the obese individual is at risk for malnutrition or is malnourished. Currently, there is no validated method for calculating caloric or protein needs for obese individuals.[74] The RDN should exercise clinical judgment when estimating nutritional needs. The nutrition plan for an obese individual with pressure injuries is to offer adequate protein and calories to promote healing rather than restricting the diet

to achieve weight loss. Once the wound has healed, the IDT can focus on a weight management program.

Neonates and Children

Critically ill neonates and children with pressure injuries and other complex wounds usually have other comorbidities that compromise their ability to consume adequate nutrients for healing.[83,84] Their increased nutrient requirement to meet normal growth needs places them at risk for pressure injury development and can slow the healing process. Early nutrition screening and assessment are important to identify malnutrition risk and implement a nutrition care plan. The EPUAP/NPIAP/PPPIA nutrition guidelines recommend as good practice conducting an age-appropriate nutrition and assessment for neonates and children at risk of pressure injury.[46] Table 10-7 illustrates the various screening tools used for this population.

Table 10-7 Pediatric Nutrition Screening and Assessment Tools

Neonates and Children Screening Tools	Evidence for Identifying Nutrition Risk	Screening (S) or Assessment Tool (A)	Clinical Setting
Subjective Global Nutritional Assessment for Children (SGNA)	No	S,A	Hospitalized children aged 1 mo to 18 y[85] (level 5) Children with cerebral palsy aged 1–12 y[86] (level 5) http://bit.ly/SGNAtool
Paediatric Nutritional Risk Score (PNRS)	No	S,A	Hospitalized neonates and children aged from birth to 16 y[87] http://bit.ly/PNRScore
Screening Tool for the Assessment of Malnutrition in Pediatrics (STAMP)	No	S,A	Hospitalized neonates and children aged from 2 to 7 y[88] Children with spinal cord injury[89] http://bit.ly/STAMPtool
Paediatric Yorkhill Malnutrition Score (PYMS)	NO	S	Hospitalized children aged 1–16 y[90] http://bit.ly/PYMStool
Screening Tool for the Risk of Impaired Nutritional Status and Growth (STRONGkids)	NO	S	Hospitalized children from birth to 17 y[91] Hospitalized children aged above 1 and not in critical care[92] http://bit.ly/STRONGkids

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Growth assessment is the strongest gauge of nutrition status in this population Anthropometric data including weight, weight/height, growth scores, head circumference, and z scores are used to determine if the neonate or child is developing according to established parameters. Z scores are preferable nutrition assessment tools since they are more descriptive than growth charts.[54,93] As part of the nutrition assessment, the RDN may conduct a nutrition-focused physical exam to determine micronutrient deficiencies and any sign of malnutrition.

A pediatric RDN should conduct a nutrition assessment weekly for critically ill children.[93,94] The RDN, pediatrician, and other members of the healthcare team should develop an individualized nutrition plan for the child. The plan should include the mode of feeding, frequent monitoring of intake and growth status, oral supplements, nutrition support, and feeding strategies for parents and caregivers to promote adequate intake of nutrients.[85,95]

Energy and protein needs for this population should be assessed frequently to avoid over- and underfeeding. Indirect calorimetry to estimate energy needs for critically ill children is recommended if available. Maintaining adequate hydration is also important for neonates and children with wounds.[95] When oral intake is not sufficient to support growth and wound healing, tube feeding should be considered if consistent with the goals of care and the caregiver. There are pediatric enteral formulas designed for children from 1 to 13 years of age. These formulas meet the DRIs for children ages 1 to 8 in 1,000 mL/d and 1,500 mL/d for children ages 9 to 14.[96] Currently there is insufficient research to make a specific recommendation for macro- and micronutrients for this age group.[93,94]

Nutrition Interventions

Daily Caloric Requirements

Estimating an individual's daily caloric requirement is part of the assessment process and assists in determining the appropriate plan for the prevention and/or treatment of wounds. Indirect calorimetry is the gold standard for measuring energy expenditure estimation and quantifies stress due to illness, injury, and other medical conditions.[97] However, indirect calorimetry is not widely used outside the acute care setting. The Harris-Benedict equation measures resting metabolic rate rather than basal energy expenditure, but controversy exists over its accuracy in obese or severely undernourished individuals. Cereda and Klersy et al.'s study noted that when estimating energy needs for individuals with pressure injuries using Harris-Benedict formula, clinicians should consider a correction factor based on the underestimation of 10% of energy needs.[98] The Mifflin–St. Jeor equation is appropriate for healthy, noninjured adults as well as overweight and obese individuals.[99,100]

The management of undernutrition is extremely important for individuals with wounds such as pressure injuries. Nutrition status, undernutrition, nutrition support, and nutritional intake are all critical to this process. The 2019/EPUAP/NPIAP/PPPIA Pressure Injury Prevention and Treatment Clinical Guidelines provide nutrition recommendations that are a resource for practitioners when developing nutrition interventions for individual patients (Table 10-8). Clinical judgment should be used when applying these guidelines, as they may not be appropriate in all circumstances.

Table 10-8 Clinical Guidelines

Recommendation with Strength of Evidence and Strength of Recommendation
4.4 Optimize energy intake for individuals at risk of pressure ulcer/injuries who are malnourished or at risk of malnutrition. **SOE = B2; Strength of Recommendation = ↑**
4.5 Adjust protein intake for individuals at risk of pressure ulcer/injuries who are malnourished or at risk of malnutrition. **GOOD PRACTICE STATEMENT**
4.6 Provide 30–35 kcal/kg body weight/d body weight for adults with a pressure injury who are malnourished or at risk of malnutrition. **SOE = B1; Strength of Recommendation = ↑**
4.7 Provide 1.25–1.5 g/kg body weight/d for adults with a pressure ulcer/injury who are malnourished or at risk of malnutrition. **SOE = B1; Strength of Recommendation = ↑↑**
4.8 Offer high-calorie, high-protein fortified foods and/or nutritional supplements in addition to the usual diet for adults who are at risk of developing a pressure ulcer/injury and who are also malnourished or at risk of malnutrition, if nutritional requirements cannot be achieved by normal dietary intake. **SOE = C; Strength of Recommendation = ↑**
4.9 Offer high-calorie, high-protein nutritional supplements in addition to the usual diet for adults with a pressure ulcer/injury who are malnourished or at risk for malnutrition, if nutritional requirements cannot be achieved by normal dietary intake. **SOE = B1; Strength of Recommendation = ↑↑**
4.10 Provide high-calorie, high-protein, arginine, zinc and antioxidant oral nutritional supplements or enteral formula for adults with a category/stage 2 or greater pressure ulcer/injury who are malnourished or at risk for malnutrition. **SOE = B1; Strength of Recommendation = ↑**
4.11 Discuss the benefits and harms of enteral or parenteral feeding to support overall health in light of preferences and goals of care with individuals at risk of pressure ulcer/injury who cannot meet their nutritional requirements through oral intake despite nutritional interventions. **GOOD PRACTICE STATEMENT**
4.12 Discuss the benefits and harms of enteral or parenteral feeding to support pressure injury treatment in light of preferences and goals of care for individuals with pressure ulcer/injury who cannot meet their nutritional requirements through oral intake despite nutritional interventions. **SOE = B1; Strength of Recommendation = ↑**
4.13 Provide and encourage adequate water intake for hydration for an individual with or at risk of a pressure ulcer/injury, when compatible with goals of care and clinical condition. **GOOD PRECTICE STATEMENT**
4.14 Conduct age-appropriate nutritional screening and assessment for neonates and children at risk of pressure ulcers/injuries. **GOOD PRACTICE STATEMENT**
4.15 For neonates and children with or at risk of pressure ulcer/injury who have inadequate oral intake, consider fortified foods, age-appropriate nutritional supplements, or enteral or parenteral nutritional support. **GOOD PRACTICE STATEMENT**

Adapted with permission from European Pressure Ulcer Advisory Panel, National Pressure Injury Advisory Panel and Pan Pacific Pressure Injury Alliance. In: E. Haesler, ed. *Prevention and Treatment of Pressure Ulcers/Injuries: Clinical Practice Guideline. The International Guideline*. EPUAP/NPIAP/PPPIA, 2019.

See Appendix “Definitions of the EPUAP NPIAP PPPIA 2019 Clinical Guideline Strengths of Evidence and Strengths of Recommendations.”

Strategies to Enhance Oral Intake

When an individual cannot meet his or her nutrition needs through consumption of traditional meals, then additional strategies should be considered. Options include providing oral supplements between meals, adding fortified foods or between-meal snacks to the meal plan. ONSs include commercial products that supply nutrients such as protein, carbohydrates, fat, vitamins, minerals, and/or conditionally essential amino acids.

Stratton et al.[101] pooled the results of four RCTs comparing ONS with routine care, following a normal diet and implementing routine pressure injury care, and one RCT comparing enteral tube feeding to routine care. The results of the meta-analysis showed that oral nutritional supplementation (primarily high protein, 400 to 500 kcal, duration of 4 to 72 weeks) was associated with a significant reduction in pressure injury development compared to routine care. A retrospective cohort study of 1,524 nursing home residents reported that the consumption of an oral supplement was a predictor of pressure injury healing.[3] Additional research as noted in the previous discussion of protein, amino acids, and micronutrients supports the value of providing ONS as a therapeutic intervention to promote healing.[49,54–57] Wilson et al. studied the timing of supplements and concluded that consuming ONS between meals resulted in better absorption of nutrients and less interference with meal intake.[102]

Fortifying foods also increases calories and nutrients; consider adding powdered milk to foods the patient is already eating, such as hot cereal or mashed potatoes. Offer small, frequent meals and snacks, such as high-calorie bars, sandwiches, Greek yogurt, and other nutrient-rich items. Enhance overall caloric and nutrient intake by tailoring meals and snacks based on the person's cultural and religious preference.[103]

Practice Point*Nutrition Supplement and Snack Schedule*

In order to provide variety and avoid flavor fatigue, it is important to offer variety and to rotate types and flavors of high-calorie/high-protein supplements and snacks. Supplements and snacks may be varied to achieve these goals, for example, offering a turkey sandwich with a glass of milk as afternoon snack and a strawberry banana shake in the evening. Rotate various types of sandwiches (chicken, turkey, tuna salad, grilled cheese, etc.) with various flavors of puddings, milkshakes, cream soups, high-protein bars, Greek yogurt, or other high-protein combinations.

Exploring why an individual isn't eating is the first step in helping to meet his or her nutritional needs. For example, is there an emotional or physical reason why eating is a problem? Find out if something is bothering the individual that is preventing him or her from eating. Provide an environment that reduces noxious smells, which may decrease appetite, while increasing pleasing aromas of food being prepared or other pleasant smells such as baking bread. A quiet, unhurried eating environment with frequent cueing is particularly helpful for cognitively impaired persons. Similarly, in evaluating the individual's physical ability to eat, consider the following questions:

Oral Intake Evaluation

- How much time does it take the individual to eat? Fatigue or fear of choking may cause the person to eat slowly.
- Does the individual have the physical ability to bring the food to the mouth? Can he or she handle eating with utensils appropriately? Neuromuscular impairments, fatigue, or decreased endurance can interfere with the individual's ability to eat independently. Consider use of assistive devices and consultation with an occupational therapist. Some may prefer "finger" foods. Even with appropriate utensils, consider whether the person has the coordination to bring the food to the mouth.
- Can the individual see the food placed before him or her? Changes in the visual field as a result of stroke, cataracts, glaucoma, or diabetes may alter the ability to see food on all or part of the meal placed before them. Arrange food so the individual can see and reach it.
- Can the individual chew? Check on the condition of the oral cavity. Provide appropriate mouth care to cleanse the mouth and stimulate the appetite. If the individual has dentures, is he or she using them and do they fit?
- Can the person swallow? Cranial nerve and other neurologic conditions can cause swallowing difficulties. Evaluate the individual for any signs of abnormal swallowing. Teach the person to direct food to the unaffected side of the mouth. Consultation from a speech language therapist for management of swallowing difficulties and recommendations about food textures may be helpful.
- Is the individual's diet unappealing and unappetizing due to the restrictions of the diet order? Diet restrictions may result in reduced food and fluid intake. Ask the RDN to assess the value of the diet restriction, and recommend individualization to a less restrictive diet, as appropriate.

Many older adults are chronically dehydrated, and ensuring adequate oral fluid intake can be challenging. Care providers who deliver trays should open and uncover all liquids and encourage individuals to

drink beverages. A frail older adult with arthritis, for example, may not be able to open sealed containers. Fluids include broth, gelatin, ice cream sorbets, sherbet, soft drinks, juice, ice cream, or any food/liquid that is fluid at room temperature. Offer additional fluids between meals, at medication pass and during activities. Use creative ways to encourage daily fluid intake. For example, give ice pops in warm climates or hot soup in cooler climates. Suggest the therapy department offer a selection of beverages after or during rehabilitation sessions.

Suggestions for meeting nutrient requirements for individuals with pressure injuries are detailed in Table 10-9.

Table 10-9 Suggestion to Meet Nutritional Needs for Pressure Injuries

All interventions must be individualized based on assessment of needs		
	High Risk (Prevention)	**Stage 1–4, Unstageable (Based on individualized assessed needs)**
Sample interventions for increased caloric needs	• Offer favorite foods • Offer fortified foods (high calorie, and protein as needed) • High-calorie med pass program • Honor food preferences • Additional interventions as needed to meet individual needs	• Offer favorite foods. Offer fortified foods (high calorie, and protein as needed) • Extra margarine, gravy, and other calorie boosters as tolerated with meals/snacks • 8 ounce half and half or whole milk (or whole milk and half and half mixed together) with meals • Fortified foods with each meal • Snacks of choice between meals • Ice cream with dinner • House supplement between meals (preferably at least 60 min before meals) • High-calorie med pass program • Additional interventions as needed to meet individual needs
Sample interventions for increased protein needs	• Extra ounce of protein per meal • 8 ounces of whole milk or high-protein milkshake each meal • High protein between meal snacks (sandwiches, cottage cheese, Greek yogurt, etc.)	• Extra ounce of protein at each meal • 8 ounces of whole milk or high protein milkshake each meal • High protein between meal snacks (sandwiches, cottage cheese, Greek yogurt, etc.) • House supplement between meals (preferably at least 60 min before meals)
Sample interventions for increased fluid needs	• 12–16 ounces favorite beverages at every meal • Offer fluid with every contact • Water or fluid of choice at the bedside	• 12–16 ounces favorite beverages at every meal • Offer fluid with every contact • Water or fluid of choice at the bedside
• **Multivitamin and mineral (up to 100% USRDI)[a]** • **Elemental zinc <40 mg daily[a]**	• Daily • NA	• Daily if intake is poor or a deficiency is confirmed or suspected • Maximum duration 2–3 wk.
• **Amino acids**		• Use a nutritional formula enriched with protein, arginine, zinc, and vitamin C. • Reevaluate as pressure ulcer heals

These are just suggestions. All interventions must be based on individual assessment of nutritional needs.

[a]Sample interventions for Vitamins/Minerals *if* deficiencies are confirmed or suspected.

Offering/Providing Enteral/Parenteral Support

Nutrition support, known as enteral or parenteral feeding, can be used to place the individual into positive nitrogen balance (in which the body maintains the same amount of protein in its tissues from day to day) according to the goals of care and whether it is compatible with the individual's and family's/surrogate's wishes. A tube feeding may be initiated when the ability to chew, swallow, and absorb nutrients through normal gastrointestinal route is compromised. This occurs in conditions such as stroke, Parkinson's disease, cancer, and dysphagia or when individuals can't meet their nutritional needs orally. Most enteral tube feeding formulas are nutritionally complete and designed for a specific purpose. Parenteral nutrition is the delivery of nutrient solutions directly into a vein, bypassing the intestine. This form of feeding is necessary for individuals when enteral tube feeding is contraindicated, is insufficient to maintain nutritional status, or has led to serious complications.

Does providing enteral feedings prevent pressure injuries? The answer may lie in the limited number of published studies involving the use of tube feedings in individuals with pressure injuries. One moderate-quality study in older hospitalized adults (*n* = 60) receiving 12 weeks of enteral nutritional support calculated to receive a mean intake of 37.9 ± 6.5 kcal/kg/d was associated with improved pressure injury size and depth compared to standard tube feeding with a mean intake of 29.1 ± 4.9 kcal/kg/d.[96]

Another study of older adults (*n* = 28), 65% of whom were tube fed, receiving 8 weeks of adequate amounts of energy (≥30 kcal/kg/d) and protein (≥1.2 g/kg/d) showed significant improvements in pressure injury surface area and PUSH score.[93] A study of older adults receiving enteral nutrition reported they had significantly more major complications (e.g., weight loss, pneumonia and death) that were deemed to be related to the intervention compared to individuals receiving an oral diet (61% vs. 34%).[104]

Palliative/End of Life/Hospice Care

Both palliative care and hospice care include a multidisciplinary approach offering comfort and support to individuals with a serious or chronic illness. The World Health Organization defines palliative care as “an approach that improves the quality of life of patients and their families facing the problem associated with life-threatening illness, through the prevention and relief of suffering by means of early identification and impeccable assessment and treatment of pain and other problems, physical, psychosocial and spiritual.”[105] Palliative care can begin at diagnosis, and can coincide with treatment. Hospice care/end of life care provides supportive care to individuals in the final phase of a terminal illness and focuses on comfort and quality of life, rather than cure. End of life/hospice care enables individuals to be comfortable and free of pain, so they can live each day as fully as possible. Hospice care begins after treatment of the disease is stopped and when it is clear that the individual is not going to survive the illness, and is usually offered when the person is expected to live 6 months or less.

The IDT has the ethical obligation to protect life and to relieve suffering; however, the individual has the right to refuse treatment, including feeding and hydration. It is the position of the Academy of Nutrition and Dietetics that “individuals have the right to request or refuse nutrition and hydration as medical treatment.”[106] Many disease conditions, such as end-stage congestive heart failure, end-stage renal failure, and advanced cancer, are considered terminal illnesses; therefore, individuals with these conditions who also have wounds are candidates for hospice care. Advanced dementia is now considered a terminal illness.[107] The IDT is responsible for discussing the risk and benefits of initiating or withdrawing EN with the individual and/or their families or surrogate. The RDN can explain that loss of appetite is common in terminally ill individuals and does not reduce quality of life. Minimizing or withholding hydration has the positive effect of reducing oral and bronchial secretions, thus relieving pulmonary congestion. Several studies of withholding nutrition indicate that the physiological

adaptation allows individuals not to suffer from the absence of nutrition or hydration.[108,109]

In a randomized controlled trial to evaluate the quality of life of advanced non–small cell lung cancer patients with early palliative care and standard oncological care, the early palliative care group had significant improvements in quality of life and fewer depressive symptoms. In addition, the early palliative care group pursued less aggressive end-of-life care and the median survival rate was longer in the early palliative care group.[110,111] Not only did the patients benefit from early palliative care, caregivers of patients with advanced cancer also showed reduced depression and stress burden from early palliative care in a randomized controlled trial.[110–112] Treatment goals should be client centered, respecting each person's unique values and personal decisions.[106]

Nursing Home Survey and Regulations: Pressure Injuries

The Center for Medicare and Medicaid Service (CMS), which regulates long-term care facilities, has targeted pressure injuries/injuries, inadequate nutrition, and unintended weight loss as key survey issues. The development of a pressure injury/injury in a person who was at low risk is automatically considered a sentinel event. Federal Tag F 686 provides guidance to surveyors as part of the CMS *State Operations Provider Certification Manual*. The guidance contains a section on Nutrition and Hydration noting that "continuing weight loss and failure of a pressure injury/injury to heal, despite reasonable efforts to improve caloric and nutrient intake, may indicate that the resident is in multisystem failure or in an end-stage or end-of-life condition warranting an additional assessment of the resident's overall condition."[113] The IDT should assist in the development of nutrition goals considering the individual's prognosis and projected clinical course (see Table 10-10).

Table 10-10 Definition of High Risk for Pressure Ulcer/Injury Development

Risk Factors

Not all risk factors are fully modifiable or can be completely addressed. Some risk factors, such as a permanent lack of sensation to an area, may not be modifiable. Some potentially modifiable risk factors, such as malnutrition or uncontrolled blood sugars, may take time to correct, despite prompt intervention. Other risk factors, such as pressure, can be modified promptly. Many studies and professional literature identify risk factors that increase a resident's susceptibility to develop or to not heal pressure PU/PIs. Examples of these risk factors include, but are not limited to

- Impaired/decreased mobility and decreased functional ability;
- Comorbid conditions, such as end-stage renal disease, thyroid disease, or diabetes mellitus;
- Drugs such as steroids that may affect healing;
- Impaired diffuse or localized blood flow, for example, generalized atherosclerosis or lower extremity arterial insufficiency;
- Resident refusal of some aspects of care and treatment;
- Cognitive impairment;
- Exposure of skin to urinary and fecal incontinence;
- Under nutrition, malnutrition, and hydration deficits; and
- The presence of a previously healed PU/PI. The history of any healed PU/PI, its origin, treatment, its stages [if known] is important assessment information, since areas of healed stage 3 or 4 PU/PIs are more likely to have recurrent breakdown.

Although the requirements do not mandate the use of any specific assessment tool (other than the RAI), many validated instruments are available to aid in assessing the risk for developing PU/PIs. It is important to keep in mind that research has shown that in a skilled nursing facility, 80% of PU/PIs develop within 2 wk of admission and 96% develop within 3 wk of admission. (Lyder, C.H., Ayello, E.A. Pressure Ulcers: A Patient Safety Issue. In: R.G. Hughes, ed. *Patient Safety and Quality: An Evidence-Based Handbook for Nurses*. Rockville, MD: Agency for Healthcare Research and Quality (US), 2008. Chapter 12. Available at: http://www.ncbi.nlm.nih.gov/books/NBK2650/)

Many clinicians utilize a standardized pressure ulcer/injury risk assessment tool to assess a resident's PU/PI risks upon admission, weekly for the first 4 wk after admission, then monthly or whenever there is a change in the resident's condition. A resident's risk may increase due to an acute illness or condition change (e.g., upper respiratory infection, pneumonia, or exacerbation of underlying congestive heart failure) and may require additional evaluation. The frequency of assessment should be based upon each resident's specific needs.

Regardless of any resident's total risk score on an assessment tool, clinicians are responsible for evaluating each existing and potential risk factor for developing a pressure injury and determining the resident's overall risk. It is acceptable if the clinician's assessment places the resident at a higher risk level than the overall score of the assessment tool based on assessment factors that are not captured by the tool. Documentation of the clinician's decision should be placed in the medical record.

Reprinted from Center for Medicare & Medicaid Services, Department of Health and Human Services. State Operations Manual Appendix PP—Guidance to Surveyors for Long Term Care Facilities (Rev. 173, 11-22-17). Available at: https://www.cms.gov/Regulations-and-Guidance/Guidance/Manuals/downloads/som107ap_pp_guidelines_ltcf.pdf. Accessed January 28, 2019.

The section on nutrition also notes: "Although some laboratory tests may help clinicians evaluate nutritional issues in a resident with pressure injury (PIs) no laboratory test is specific or sensitive enough to warrant serial/repeated testing." A practitioner may order test(s) that provide useful additional information or help with management of treatable conditions at their discretion. CMS notes, "Not all risk factors are fully modifiable or can be completely addressed. Some risk

factors, such as a permanent lack of sensation to an area, may not be modifiable. Some potentially modifiable risk factors, such as malnutrition or uncontrolled blood sugars, may take time to correct, despite prompt intervention." Many clinicians utilize a standardized pressure injury/injury risk assessment tool to assess a resident's PI risks upon admission, weekly for the first 4 weeks after admission, then monthly or whenever there is a change in the resident's condition.

Research on the value of the Braden Nutrition Subscale to evaluate nutrition risk was a secondary data analysis from the TURN for Injury Reduction (TURN) study of 690 nursing home residents in the United States and Canada.[113] Repeated weekly measures of the components of the subscale: percentage of meals consumed, mean number and sources of protein servings, estimated percentage of supplements and snack consumed, and incidence of new pressure injuries were collected plus weight status for residents who lived in the facility at least 3 months and had intact skin. The certified nursing assistants (CNA) reported that 59.2% of residents at high pressure injury risk and 61.9% of those at moderate risk consumed ≤75% of most meals. The authors concluded that meal intake of the residents who were at high or moderate risk for pressure injury development tended to match the intake ratings defined in the nutrition subscale.[114] There was also a relationship between the activity subscale and meal intake. Residents with changes in mobility corresponded with those who had decreased intake. The results also offered information on hydration status and adequacy of total meal intake, including protein and nutrient supplementation. Careful and frequent completion and monitoring of the Braden nutrition subscale can serve as a source of preliminary screening information that offers an awareness into meal completion patterns.[114]

Documentation in the Medical Record

Medical nutrition therapy documentation in the medical record should include:

- amount of food consumed in both quantity (percentage of meal served) and quality (type of food) related to amount needed
- average fluid consumed with meals
- amount in ounces of ONSs or percentage of snacks consumed
- ability to eat—assisted, supervised, or independent
- acceptance or refusal of diet, meals, or supplements
- current weight and percentage gained or lost
- new conditions affecting nutritional status, such as introduction of thickened liquids or new diagnosis
- new medications affecting nutritional status
- current laboratory results, if appropriate
- wound condition, stage, and progress toward healing
- current calorie, protein, or fluid requirements
- monitoring and evaluation of care plan including revisions
- education of the individual and/or family concerning the importance of nutrition interventions for healing and improving nutritional status.

Summary

Nutrition is an important consideration when treating an individual with pressure injuries, chronic wounds, or diabetic injuries. Early nutrition screening and assessment are key elements for diagnosing malnutrition, which can impede healing. Nutrition not only facilitates healing but also improves or stabilizes the individual's quality of life. The focus should be on optimal nutrition for each individual, which for some persons may be achieved by a diet that includes ONSs or fortified foods and allows the individual to enjoy his or her favorite foods. For others, enteral and parenteral nutrition support may be necessary. The amount and type of nutrition support provided to individuals with pressure injuries should be consistent with both the medical goals and the individual's wishes.

PATIENT SCENARIO

Clinical Data

MT is an 81-year-old woman admitted to a rehabilitation center following surgery 4 days ago for a hip fracture. She has a stage 4 pressure injury on her coccyx that she acquired while lying on her kitchen floor for several hours after she fell. Additional diagnosis includes dementia, hypertension, and cardiovascular disease with history of MI. Her height is 644 inches and usual body weight is 149 pounds. The medical record does not include weight PTA, and the family cannot provide that information. However, the family states she was surviving on bananas and granola bars with one good meal per day provided by family. Her intake is fair (50%) of her low-fat/low-cholesterol, 2-g sodium diet (which provides approximately 2,000 kcals/d). She chooses not to eat beef or pork, and likes to snack on cookies and other sweets between meals. Her MNA screening score is 7 indicating malnutrition, and she has been referred to the RDN for a nutritional assessment.

Her biochemical data include H/H 10.8/33.1, Glu 108, Alb 3.1, Na 137, K+ 4.5, BUN 18, Creat 0.7, Chol 160. Medications include beta-blocker, statins, diuretic, calcium, vitamin D, and pain medications. The physical therapy department is working to improve her mobility but notes her grip strength is poor, a possible indication of malnutrition. The wound care team is addressing the appropriate treatment and support surfaces for her pressure injury.

Clinical Malnutrition

Acute injury–associated malnutrition (hip surgery and pressure injury) and chronic disease–associated malnutrition.

History and Clinical Data

MNA nutritional screen indicated malnutrition. Chronic inflammation associated with CVD, plus current decline in food/fluid intake and historical diet of poor quality prior to admission per interview with family. Hip surgery and current stage 4 pressure injury associated with acute inflammatory response compounded by comorbidities.

Anthropometric Data

BMI 25.6 is only slightly above normal range; unsure of weight loss prior to admission, but at current rate of intake unintended weight loss is likely without intervention.

Laboratory Data

Low H&H possibly related to long-standing poor diet and recent fracture and surgery. Low serum albumin due to chronic and acute inflammation.

Dietary Intake

MT’s history and current intake indicate poor quality. She also expressed poor appetite due to pain. Her diet has been of poor nutritional quality, including a lack of high–biological value protein, nutrient-rich sources of carbohydrates, zinc, and vitamin C—all of which are required to heal her pressure injury.

Functional Outcome

Physical therapy notes her poor endurance during rehabilitation sessions, and her grip strength is poor for age and gender.

Nutritional Diagnosis

Severe malnutrition in context of acute injury and chronic poor diet as evidenced by reduced energy intake for 7 days and reduced grip strength for age and gender.

Nutritional Intervention

The RDN and MT worked together to develop a selective menu plan to achieve her estimated caloric needs with a focus on high-quality protein and healthy carbohydrate choices for each meal. Per RDN's recommendation, the physician ordered a regular diet and 30 mL of a concentrated high-kcal/ high-protein oral nutritional supplement four times a day with med pass. The supplement has 100 kcal and 17 g of protein plus vitamin C, zinc, and arginine, which provides 34 g of protein in addition to the approximately 80 g in her meals (at 100% intake). MT increased her fluid intake to at least 8 cups of fluid daily, and her intake improved as her pain subsided. The physician ordered routine labs to check her H&H levels and renal function to ensure high level of protein was tolerated. Her weight was monitored weekly until stable.

Show What You Know

1. **MT weighs 149 pounds and has a stage 4 pressure injury and a poor appetite; her recommended daily protein requirement is:**
   A. 102 to 135 g.
   B. 88 to 107 g.
   C. 85 to 102 g.
   D. 68 to 86 g.

2. **MT only consumes 50% of her low-fat/low-cholesterol, 2 g sodium diet. She complains the food has no flavor and is at risk for unintended weight loss, and her pressure injury is not healing. The wound care team should:**
   A. request that nursing weigh MK daily for the next 2 weeks or until weight is stable.
   B. request RDN referral to assess her nutritional status and ask the physician to consider ordering a regular diet.
   C. request the physician order a high-calorie supplement three times a day.
   D. request the physician order zinc sulfate 220 mg three times a day.

3. **MT has a large, draining stage 4 pressure injury, and her daily caloric requirements are estimated at 2,200 kcal. She is unable to consume this amount of calories in three meals each day. She should be offered:**
   A. a high-calorie, high-protein supplement with all her meals.
   B. double portions of protein at each meal and a shake at bedtime.
   C. high-protein, high-calorie supplement with arginine and micronutrients between meals.
   D. a high-calorie/high-protein supplement at breakfast and three times a day.

4. **Which of the following 2019 EPUAP//NPIAP/PPPIA nutrition guidelines for treatment is correct?**

   A. Offer 500 mg ascorbic acid daily to adults with a pressure injury.

   B. Offer 30 to 35 kcal/kg body weight daily for adults with a pressure injury.

   C. Offer 25 mg elemental zinc and 500 mg ascorbic acid twice daily.

   D. Offer 1.25 to 2.0 g protein/kg body weight for adults with a pressure injury.

5. **Which of the following amino acids are considered conditionally indispensable during periods of stress?**

   A. Arginine and glutamine

   B. Alanine and glutamine

   C. Valine and arginine

   D. Lysine and glutamic acid

Answers to these questions are found on page 683.

References

1. Frykberg, R.G., Banks, J. “Challenges in the Treatment of Chronic Wounds.” Advances in Wound Care 4(9):560-82, 2015.
2. Litchford, M.D., Dorner, B., Posthauer, M.E. “Malnutrition as a Precursor of Pressure Ulcers.” Advances in Wound Care 3(1):54-63, 2014.
3. Bergstrom, N., Horn, S.D., Smout, R.J., et al. “The National Pressure Injury Long-Term Care Study: Outcomes of Pressure Injury Treatments in Long-term Care.” Journal of the American Geriatrics Society 53:1721-9, 2005.
4. Fry, D.E., Pine, M., Jones, B.L., et al. “Patient Characteristics and the Occurrence of Never Events.” Archives of Surgery 145(2):148-51, 2010.
5. Shahin, E.S., Meijers, J.M., Schols, J.M., et al. “The Relationship Between Malnutrition Parameters and Pressure Ulcers in Hospitals and Nursing Homes.” Nutrition 26(9):886-9, 2010.
6. Banks, M., Bauer, J., Graves, N., et al. “Malnutrition and Pressure Ulcer Risk in Adults in Australian Health Care Facilities.” Nutrition in Clinical Practice 26(9):896-901, 2010.
7. Iizaka, S., Okuwa, M., Sugama, J., et al. “The Impact of Malnutrition and Nutrition-related Factors on the Development and Severity of Pressure Ulcers in Older Patients Receiving Home Care.” Clinical Nutrition 29(1):47-53, 2010.
8. Edsberg, L.E., Langemo, D., Baharestani, M.M., et al. “Unavoidable Pressure Injury: State of the science and consensus conference.” Journal of Wound, Ostomy and Continence Nursing 41(4):313-34, 2014.
9. Barker, L., Gout, B., Crowe, T. “Hospital Malnutrition: Prevalence, Identification and Impact on Patients and the Healthcare System.” International Journal of Environmental Research and Public Health, 8:514-21, 2011.
10. Törma, J., Winblad, U., Cederholm, T., et al. “Does Undernutrition Still Prevail Among Nursing Home Residents?” Clinical Nutrition 32:562, 2013.
11. White, J., Guenter, P., Jensen, G., et al. “Consensus statement of the Academy of Nutrition and Dietetics/American Society for Parenteral and Enteral Nutrition: Characteristics Recommended for the Identification and Documentation of Adult Malnutrition (Undernutrition).” Journal of the Academy of Nutrition and Dietetics 112:730-8, 2012.
12. Kane, R.L., Talley, K.M., Shamliyan, T., et al. Common Syndromes in Older Adults Related to Primary and Secondary Prevention. Evidence Report/Technology Assessment No. 87. AHRQ Publication No. 11-05157-EF-1. Rockville, MD: Agency for Healthcare Research and Quality, July 2011.
13. Dorland’s Illustrated Medical Dictionary, 32nd ed. New York, NY: Elsevier Health sciences Division, 2011.

14. Jensen, G.L., Mirtallo, J., Compher, C., et al. “Adult Starvation and Disease-Related Malnutrition: A Rational Approach for Etiology-Based Diagnosis in the Clinical Practice Setting from the International Consensus Guidelines Committee.” Journal of Parenteral and Enteral Nutrition 34(2):165-9, 2010.
15. Demling, R.H. “Nutrition, Anabolism, and the Wound Healing Process: An Overview.” Eplasty 9: 65-94, 2009.
16. Cederholm, T., Bosaeus, I., Barazzoni, R., et al. “Diagnostic Criteria for Malnutrition: An ESPEN Consensus Statement.” Clinical Nutrition 34:335-40, 2015.
17. The Joint Commission. Nutritional and Functional Screening-Requirement. The Joint Commission, 2016. Available at https://www.jointcommission.org/mobile/standards_information/jcfaqdetails.aspx?StandardsFAQId=1604&StandardsFAQChapterId=29&ProgramId=0&ChapterId=0&IsFeatured=False&IsNew=False&Keyword=
18. Thomas, D.R. “Nutritional Assessment in Long Term Care.” Nutrition in Clinical Practice 23:383-7, 2008.
19. Kaiser, M.J., Bauer, J.M., Ramsch, C., et al. “Validation of the Mini Nutritional Assessment Short-Form (MNA-SF): A Practical Tool for Identification of Nutritional Status.” Journal of Nutrition, Health & Aging 13(9):782-8, 2009.
20. Hengstermann, S.A., Fischer, A., Steinhagen-Thiessen, E., et al. “Nutrition Status and Pressure Injury: What We Need for Nutrition Screening.” Journal of Parenteral and Enteral Nutrition 31(4):288-94, 2007.
21. Tsai, A.C., Chang, T.L., Wang, Y.C., et al. “Population-Specific Short-Form Mini Nutritional Assessment with Body Mass Index or Calf Circumference Can Predict Risk of Malnutrition in Community-Living or Institutionalized a People in Taiwan.” Journal of the American Dietetic Association 110(9):1328-34, 2009.
22. Grattagliano, I., Marasciulo, I., Paci, C., et al. “The Assessment of the Nutritional Status Predicts the Long-term Risk of Major Events in Older Adults.” European Geriatric Medicine 8:273-4, 2017.
23. Corrales J.M., Gayo, N.P., Águila, M.P., et al. “The Importance of Using a Nutritional Risk Analysis Scale in Patients Admitted to Continued Care.” Journal of the European Wound Management Association 14(2):27-31, 2014.
24. Poulia, K.A., Yannakoulia, M., Karageorgou, D., et al. “Evaluation of the Efficacy of Six Nutritional Screening Tools to Predict Malnutrition in the Elderly.” Clinical Nutrition 31(3):378-85, 2012.
25. Tsaousi, G., Stavrou, G., Ionnidis, A., et al. “Pressure Ulcers and Malnutrition: Results for a Snapshot Sampling in a University Hospital.” Medical Principles and Practice 24:11-6, 2015.
26. Kondrup, J., Rasmussen, H.H., Hamberg, I., et al. “Nutritional Risk Screening (NRS 2002): A New Method Based on an Analysis of Controlled Clinical Trials.” Clinical Nutrition 13:321-36, 2003.

27. Neelemant, F., Kruizenga, H.M., de Vet, H.C., et al. “Screening Malnutrition in Hospital Outpatients. Can the SNAQ Malnutrition-Screening Tool also Be Applied to This Population?” Clinical Nutrition 27(3):439-46, 2008.
28. Academy of Nutrition and Dietetics. Abridged Nutrition Care Process Terminology (NCPT) Reference Manual: Standardized Terminology for the Nutrition Care Process. Chicago, IL. Academy of Nutrition and Dietetics, 2018.
29. The Academy of Nutrition and Dietetics. Unintended Weight Loss (UWL) in Older Adults Evidence-Based Nutrition Practice Guideline. Chicago, IL: American Dietetic Association (ADA), 2009.
30. Thomas, D.R. “Anorexia: Aetiology, Epidemiology, and Management in the Older People.” Drugs & Aging 26:557-70, 2009.
31. Pronsky, Z.M. Food Medication Interactions, 19th ed. Birchrunville, PA: Food-Medication Interactions, 2018.
32. Jensen, G.I., Bistrian, B., Roubenoff, R., et al. Malnutrition Syndromes: A Conundrum vs Continuum. Journal of Parenteral and Enteral Nutrition 33(6):710-6, 2009.
33. Academy of Nutrition and Dietetics Evidence Analysis Library. Does Serum Albumin Correlate With Weight Loss in Four Models of Prolonged Protein Energy Restriction: Anorexia Nervosa, Non-Malabsorptive Gastric Partitioning Bariatric Surgery, Calorie-Restricted Diets or Starvation. Chicago, IL: Academy of Nutrition and Dietetics, 2009. Available at: https://www.andeal.org/topic.cfm?cat=4302&template=sort_list_template&key=2721
34. Myron Johnson, A., Merlini, G., Sheldon, J., et al. “Clinical Indications for Plasma Protein Assays: Transthyretin (Prealbumin) in Inflammation and Malnutrition.” Clinical Chemistry and Laboratory Medicine 45(3):419-26, 2007.
35. Fuhrman, M.P., Charney, P., Mueller, C.M. “Hepatic Proteins and Nutrition Assessment.” Journal of the American Dietetic Association 104(8):1258-64, 2004.
36. Lim, S.H., Lee, J.S., Chae, S.H., et al. “Prealbumin Is Not a Sensitive Indicator of Nutrition and Prognosis in Critically Ill Patients.” Yonsei Medical Journal 46(1):21-6, 2005.
37. Shenkin, A. “Serum Prealbumin: Is It a Marker of Nutritional Status or of Risk of Malnutrition.” Clinical Chemistry 52(12):2281-5, 2006.
38. Robinson, M.K., Trujillo, E.B., Mogensen, K.M., et al. “Improving Nutritional Screening of Hospitalized Patients: The Role of Prealbumin.” Journal of Parenteral and Enteral Nutrition 27(6):389-96, 2003.
39. Litchford, M.D. Laboratory Assessment of Nutritional Status: Bridging Theory and Practice. Greensboro, NC: Case Software & Books, 2017.
40. Dorner, B. Diet and Nutrition Care Manual. Dunedin, FL: Becky Dorner & Associates, Inc., 2019.

41. Center for Medicare & Medicaid Services, Department of Health and Human Services. State Operations Manual, Appendix PP—Guidance to Surveyors for Long Term Care Facilities (Rev.173, 11-22-17). Available at: https://www.cms.gov/Regulations-and-Guidance/Guidance/Manuals/downloads/som107ap_pp_guidelines_ltcf.pdf
42. Bernstein, M., Munoz, N. Nutrition for the Older Adult, 2nd ed. Burlington, MA: Jones & Barlett Learning, 2019
43. American Medical Directors Association. Dehydration and Fluid Maintenance Clinical Practice Guideline. Columbia, MD: AMDA, 2009.
44. Corrigan, M.I. Clinical: Water, Electrolytes, and Acid-Base Balance. In: L.K. Mahan, J.L. Raymond. Krause's Food and the Nutrition Care Process, 14th ed. St. Louis, MO: Elsevier, 2017:85-97.
45. The National Heart, Blood and Lung Institute. Aim for a Healthy Weight. Available at: https://www.nhlbi.nih.gov/health/educational/lose_wt/risk.htm
46. European Pressure Injury Advisory Panel, National Pressure Injury Advisory Panel and Pan Pacific Pressure Injury Alliance. In: E. Haesler, ed. Prevention and Treatment of Pressure Ulcers: Clinical Practice Guideline. 2019.
47. Mueller, C.M. The A.S.P.E.N. Adult Nutrition Support Core Curriculum, 3rd ed. Silver Springs, MD: American Society for Enteral and Parenteral Nutrition, 2017.
48. Lee, S.K., Posthauer, M.E., Dorner, B., et al. "Pressure Injury Healing with a Concentrated, Fortified, Collagen Protein Hydrolysate Supplement: A Randomized Controlled Trial." Advances in Skin & Wound Care 19(2):92-6, 2006.
49. Ohura, T., Nakajo, T., Okada, S., et al. "Evaluation of Effects of Nutrition Intervention on Healing of Pressure Ulcers and Nutritional States (Randomized Controlled Trial)." Wound Repair and Regeneration 19(3):330-6, 2011.
50. Wolfe, R.R., Miller, S.L. "The Recommended Dietary Allowance of Protein: A Misunderstood Concept." Journal of the American Medical Association 299(24):2891-3, 2008.
51. Campbell, W.W., Johnson, C.A., McCabe, C., et al. "Dietary Requirements of Younger and Older Adults." The American Journal of Clinical Nutrition 88:1332-9, 2008.
52. Bauer, J.G., Biolo, G., Cederholm, T., et al. "Evidence-based Recommendations for Optimal Protein Intake for Older People: A Position Paper form the PROT-AGE Study Group." Journal of the American Medical Directors Association 14(8):542-59, 2013.
53. Barbul, A., Lazarou, S.A., Efron, D.T., et al. "Arginine Enhances Wound Healing and Lymphocyte Immune Response in Humans." Surgery 108(2):331-7, 1990.
54. Cereda, E.A., Gini, A., Pedrolli, C., et al. "Disease-specific, Versus Standard, Nutritional Support for the Treatment of Pressure Ulcers in Institutionalized

Older Adults: A Randomized Controlled Trial." Journal of the American Geriatrics Society 57(8):1395-402, 2009.
55. Cereda, E., Klersy, C., Andreola, M., et al. "Cost-effectiveness of a Disease-Specific Oral Nutritional Support for Pressure Injury Healing." Clinical Nutrition 36(1):246-52, 2017.
56. van Anholt, R.D., Sobotka, L., Meijer, E.P., et al. "Specific Nutritional Support Accelerates Pressure Injury Healing and Reduces Wound Care Intensity in Non-Malnourished Patients." Nutrition 26(9): 867-72, 2010.
57. Desneves, K.J., Todorovic, B.E., Cassar, A., et al. "Treatment with Supplementary Arginine, Vitamin C and Zinc in Patients with Pressure Ulcers: A Randomised Controlled Trial." Clinical Nutrition 24(6):979-87, 2005.
58. Arnold, M., Barbaul, A. "Nutrition and Wound Healing." Plastic and Reconstructive Surgery 117(7 Suppl):42S-58S, 2006.
59. Hobson, R. "Vitamin E and Wound Healing: An Evidence-based Review." International Wound Journal 13(3):331-5, 2016.
60. Ronchetti, I.P., Quaglino, D., Bergamini, G. Ascorbic Acid and Connective Tissue. Subcellular Biochemistry. Volume 25: Ascorbic Acid: Biochemistry and Biomedical Cell Biology. New York: Plenum Press, 1996.
61. ter Riet, G., Kessels, A.G., Knipschild, P.G.. "Randomized Clinical Trial of Ascorbic Acid in the Treatment of Pressure Ulcers." Journal of Clinical Epidemiology 48(12):1453-60, 1995.
62. Hambidge K.M., Krebs N.F., "Zinc Deficiency: A Special Challenge." The Journal of Nutrition 137:1101-5, 2007.
63. Norris, J.R., Reynolds, R.E. "The Effect of Oral Zinc Sulfate Therapy on Decubitus Ulcers." Journal of the American Geriatrics Society 19:793-7, 1971.
64. Institute of Medicine (US) Panel on Micronutrients. Dietary Reference Intakes (DRIs): Estimated Average Requirements Food and Nutrition Board, Dietary Reference Intakes for Vitamin A, Vitamin K, Arsenic, Boron, Chromium, Copper, Iodine, Iron, Manganese, Molybdenum, Nickel, Silicon, Vanadium, and Zinc. Washington, DC. National Academy Press, 2001.
65. Willis, M.S., Monaghan, S.A., Miller M.L., et al. "Zinc Induced Copper Deficiency: A Report of Three Cases Initially Recognized on Bone Marrow Examination." The American Journal of Clincial Pathology 123:125-31, 2005.
66. Malone, A., Russell, M.K. Nutrient Requirements. In: P. Charney, A. Malone, eds. Academy of Nutrition and Dietetics Pocket Guide to Nutrition Assessment. Chicago, IL: Academy of Nutrition and Dietetics, 2016.
67. Academy of Nutrition and Dietetics Nutrition Care Manual. Available at: http://www.nutritioncaremanual.org
68. Niedert K. Nutrition Assessment of Older Adults. In: Niedert K, ed. Nutrition Care of the Older Adult: A Handbook for Nutrition Throughout the Continuum of Care. Chicago, IL: Academy of Nutrition and Dietetics, 2016:73-106.

69. Elliot CH, ed. Dietetics in Health Care Communities Pocket Resource for Nutrition Assessment, 2017 ed. Chicago, IL: Academy of Nutrition and Dietetics, 2017.
70. Institute of Medicine, National Academy of Sciences. Dietary Reference Intakes for Water, Potassium, Sodium, Chloride, and Sulfate. Washington, DC: National Academy Press, 2004.
71. Molnar, J.A., Underdown, M.J., Clark, W.A. “Nutrition and Chronic Wounds.” Advances in Wound Care 3(11):664-81, 2014.
72. Molnar, J.A., Vlad, L.G., Gumus, T. “Nutrition and Chronic Wounds: Improving Clinical Outcomes.” Plastic and Reconstructive Surgery 138(3 Suppl):71S-81S, 2016.
73. Huckleberry, Y. “Nutritional Support and the Surgical Patient.” American Journal of Health-System Pharmacy 61(7):671-84, 2004.
74. Posthauer, M.E., Banks, M., Dorner, B., et al. “The Role of Nutrition for Pressure Injury Management: National Pressure Injury Advisory Panel, European Pressure Injury Advisory Panel, and Pan Pacific pressure Injury Alliance White Paper.” Advances in Skin & Wound Care 28(4):175-88, 2015.
75. Lioupis, M.D. “Effects of Diabetes Mellitus on Wound Healing: An Update.” Journal of Wound Care 14(2):84-6, 2005.
76. Marston, W.A.; Dermagraft Diabetic Foot Injury Study Group. “Risk Factors Associated with Healing Chronic Diabetic Foot Ulcers: The Importance of Hyperglycemia.” Ostomy/Wound Management 52(3):26-8, 2006.
77. Smart H, AlGhareeb AM, Smart SA. 25-Hydroxyvitamin D Deficiency: Impacting Deep-wound Infection and Poor Healing Outcomes in Patients with Diabetes. Advances in Skin & Wound Care 32(7);321-8, 2019.
78. Evert, A.B., Boucher, J.L., Cypress, M., et al. “Nutrition Therapy Recommendations for the Management of Adults with Diabetes.” Diabetes Care 37(1):120-43, 2014.
79. Dorner, B., Friedrich, E.K. “Position of the Academy of Nutrition and Dietetics: Individualized Nutrition Approaches for Older Adults: Long Term Care, Post-Acute Care and Other Setting.” Journal of the Academy of Nutrition and Dietetics 118(4):724-35, 2018.
80. Delmore, B., Cohen, J.M., O’Neill, D., et al. “Reducing Postsurgical Wound Complications: A Critical Review.” Advances in Skin & Wound Care 30(6):272-85, 2017.
81. World Health Organization. Obesity. Available at: http://www.afro.who.int/health-topics/obesity
82. Kim, J.J., Rogers, A.M., Ballem, N., et al. “ASMBS Updated Position Statement on Insurance Mandated Preoperative Weight Loss Requirements.” Surgery for Obesity and Related Diseases 12(5):955-9, 2016.
83. Mehta N, Compher C, A.S.P.E.N. Board of Directors. A.S.P.E.N. Clinical Guidelines: Nutrition Support of the Critically Ill Child. Journal of Parenteral and Enteral Nutrition 33(3):260-76, 2009.

Secker, D.J., Jeejeebhoy, K.N. Subjective Global Nutritional Assessment for
084. children. The American Journal of Clinical Nutrition 85(4):1083-9, 2007.
85. Minocha, P., Sitaraman, S., Choudhary, A., et al. Subjective Global Nutritional Assessment: A Reliable Screening Tool for Nutritional Assessment in Cerebral Palsy Children. Indian Journal of Pediatrics 85(1):15-9, 2018.
86. White, M., Lawson, K., Ramsey, R., et al. Simple Nutrition Screening Tool for Pediatric Inpatients. Journal of Parenteral and Enteral Nutrition 40(3): 392-8, 2016.
87. McCarthy, H., Dixon, M., Crabtree, I., et al. The Development and Evaluation of the Screening Tool for Assessment of Malnutrition in Paediatrics (STAMP) for Use by Healthcare Staff. Journal of Human Nutrition and Dietetics 25:311-8, 2012.
88. Wong, S., Graham, A., Hirani, S.P., et al. Validation of the Screening Tool for the Assessment of Malnutrition in Paediatrics (STAMP) in patients with spinal cord injuries (SCIs). Spinal Cord 51(5):424-9, 2013.
89. Lestari, N.E., Nurhaeni, N., Wanda, D. The Pediatric Yorkhill Malnutrition Score Is a Reliable Malnutrition Screening Tool. Comprehensive Child and Adolescent Nursing 40(Suppl 1):62-8, 2017.
90. Huysentruyt, K., Alliet, P., Muyshont, L., et al. The STRONG(kids) Nutritional Screening Tool in Hospitalized Children: A Validation Study. Nutrition 29(11-12):1356-61, 2013.
91. Hulst, J.M., Zwart, H., Hop, W.C., et al. Dutch National Survey to Test the STRONGkids Nutritional Risk Screening Tool in Hospitalized Children. Clinical Nutrition 29(1):106-11, 2010.
92. Joosten, K.F., Hulst, J.M. Nutritional Screening Tools for Hospitalized Children: Methodological Considerations. Clinical Nutrition 33(1):1-5, 2014.
93. Mehta, N.M., Skillman, H.E., Irving, S.Y., et al. Guidelines for the Provision and Assessment of Nutrition Support Therapy in the Pediatric Critically Ill Patient: Society of Critical Care Medicine and American Society for Parenteral and Enteral Nutrition. Journal of Parenteral and Enteral Nutrition 41(5):706-42, 2017.
94. Rodriguez-Key, M., Alonzi, A. Nutrition, Skin Integrity, and Pressure Injury Healing in Chronically Ill Children: An Overview. Ostomy/Wound Management 53(6):56-62, 2007.
95. Malone, A., Carney, L.N., Mays, A., eds. ASPEN Enteral Nutrition Handbook, 2nd ed. Silver Springs, MD: American society for Parenteral and Enteral Nutrition, 2019.
96. Schoeller, D. “Making Indirect Calorimetry a Gold Standard for Predicting Energy Requirements for Institutionalized Patients.” Journal of the American Dietetic Association 107(3):390-2, 2007.
97. Cereda, E., Klersy, C., Rondanelli, M., et al. “Energy Balance in Patients with Pressure Ulcers: A Systematic Review and Meta-Analysis of Observational Studies.” Journal of the American Dietetic Association 111:1868-76, 2011.

98. Mifflin, M.D., St Jeor, S.T., Hill, L.A. et al. “A New Predictive Equation for Resting Energy Expenditure in Healthy Individuals.” American Journal of Clinical Nutrition 51(2):241-7, 1990.
99. Frankenfield, D., Roth-Yousey, L., Compher, C. “Comparison of Predictive Equations for Resting Metabolic Rate in Healthy Nonobese and Obese Adults: A Systematic Review.” Journal of the American Dietetic Association 105:775-89, 2005.
100. Stratton, R.J., Ek, A.C., Engfer, M., et al. “Enteral Nutritional Support in Prevention and Treatment of Pressure Ulcers: A Systematic Review and Meta-analysis.” Ageing Research Reviews 4(3):422-50, 2005.
101. Wilson, M.M., Purushothaman, R., Morley, J.E. “Effect of Liquid Dietary Supplements on Energy Intake in the Elderly.” The American Journal of Clinical Nutrition 75(5):944-7, 2002.
102. Collins, N., Friedrich, L., Posthauer, M.E. “The Nutritional Melting Pot: Understand the Influence of Food, Culture, and Religion on Nutrition Interventions for Wound Healing.” Ostomy/Wound Management 55(5):14-21, 2009.
103. Arinzon, Z., Peisakh, A., Berner, Y.N. “Evaluation of the Benefits of Enteral Nutrition in Long-Term Care Elderly Patients.” Journal of the American Medical Directors Association 9(9):657-62, 2008.
104. World Health Organization. WHO Definition of Palliative Care. Available at: https://www.who.int/cancer/palliative/definition/en/
105. Mallet, J.O., Schwartz, D.B., Posthauer, M.E. “Ethical and Legal Issues in Feeding and Hydration.” Journal of the Academy of Nutrition and Dietetics 113(6):828-33, 2013.
106. Volicer, L. “Goals of Care in Advanced Dementia: Quality of Life, Dignity, and Comfort.” The Journal of Nutrition, Health & Aging 11(6):481, 2007.
107. Raijmakers, N.J., van Zuylen, L., Costantini, M., et al. “Artificial Nutrition and Hydration in the Last Week of Life in Cancer Patients. A Systematic Literature Review of Practices and Effects.” Annals of Oncology 22(7):1478-86, 2011.
108. Casareett, D., Kapo, J., Caplan, A. “Appropriate Use of Artificial Nutrition and Hydration—Fundamental Principles and Recommendations.” New England Journal of Medicine 353(24):2607-12, 2005.
109. Temel, J.S., Greer, J.A., Muzikansky, A., et al. “Early Palliative Care for Patients with Metastatic Non-Small-Cell Lung Cancer.” New England Journal of Medicine 363(8):733-42, 2010.
110. Dionne-Odom, J.N., Azuero, A., Lyons, K.D., et al. “Benefits of Early Versus Delayed Palliative Care to Informal Family Caregivers of Patients with Advanced Cancer: Outcomes From the ENABLE III Randomized Controlled Trial.” Journal of Clinical Oncology 33(13):1446-52, 2015.
111. Boyce, B. “An Ethical Perspective on Palliative Care.” Journal of the Academy of Nutrition and Dietetics 117(6): 970-2, 2017.

112. Centers for Medicare and Medicaid Services. State Operations Manual: Appendix PP Guidance to Surveyors for Long Term Care Facilities. Retrieved December 11, 2017. Available at: https://www.cms.gov/Medicare/Provider-Enrollment-and-Certification/GuidanceforLawsAndRegulations/Downloads/Appendix-PP-State-Operations-Manual.pdf. Accessed on January 7, 2020.
113. Kennerly, S., Boss, L., Yap, T.L., et al. “Utility of Braden Scale Nutrition Subscale Rating as an Indicator of Dietary Intake and Weight Outcomes among Nursing Home Residents at Risk for Pressure Ulcers.” Healthcare(Basel) 3(4):879-97, 2015.

11

Pressure Redistribution: Seating, Positioning, and Support Surfaces

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Objectives

After completing this chapter, you'll be able to:

- discuss tissue biomechanical properties, their measurement, and their relationship to soft tissue–loading tolerance
- identify support surface performance characteristics related to the maintenance of tissue integrity
- describe the features, functions, and limitations of various support surfaces
- outline an assessment process for selecting an appropriate support surface and related interventions (e.g., positioning and repositioning).

This chapter is written to describe support surfaces and to relate those descriptions to patients' needs for pressure injury prevention and treatment. Following a brief introduction on preventing pressure injuries is a section on soft tissue biomechanics that describes the structure and behavior of soft tissue related to its interaction with support surfaces. This is fundamental information needed to understand how support surfaces may help or hinder a patient. The next section includes information on support surface characteristics. These characteristics describe support surface interactions with the patient. Next the chapter includes descriptions of materials, components and features of support surfaces. These elements represent the current state of the art in support surface products. Finally, the chapter ties the myriad of tissue, feature, and device information to the needs of patients to assist in good clinical decision-making concerning choosing and using support surfaces. One patient scenario is included for illustration.

Preventing Pressure Injuries

Multiple intervention strategies are needed to prevent and treat pressure injuries. Managing loads on the skin and associated soft tissue is one of these strategies. A comprehensive care plan should include pressure redistribution and microclimate control strategies for individuals both while in bed and when seated. Properly chosen support surfaces; adequate periodic pressure redistribution; protection of especially vulnerable bony prominences, such as the heels, sacrum, and coccyx; and consideration of special patient needs are all essential components of the care plan.

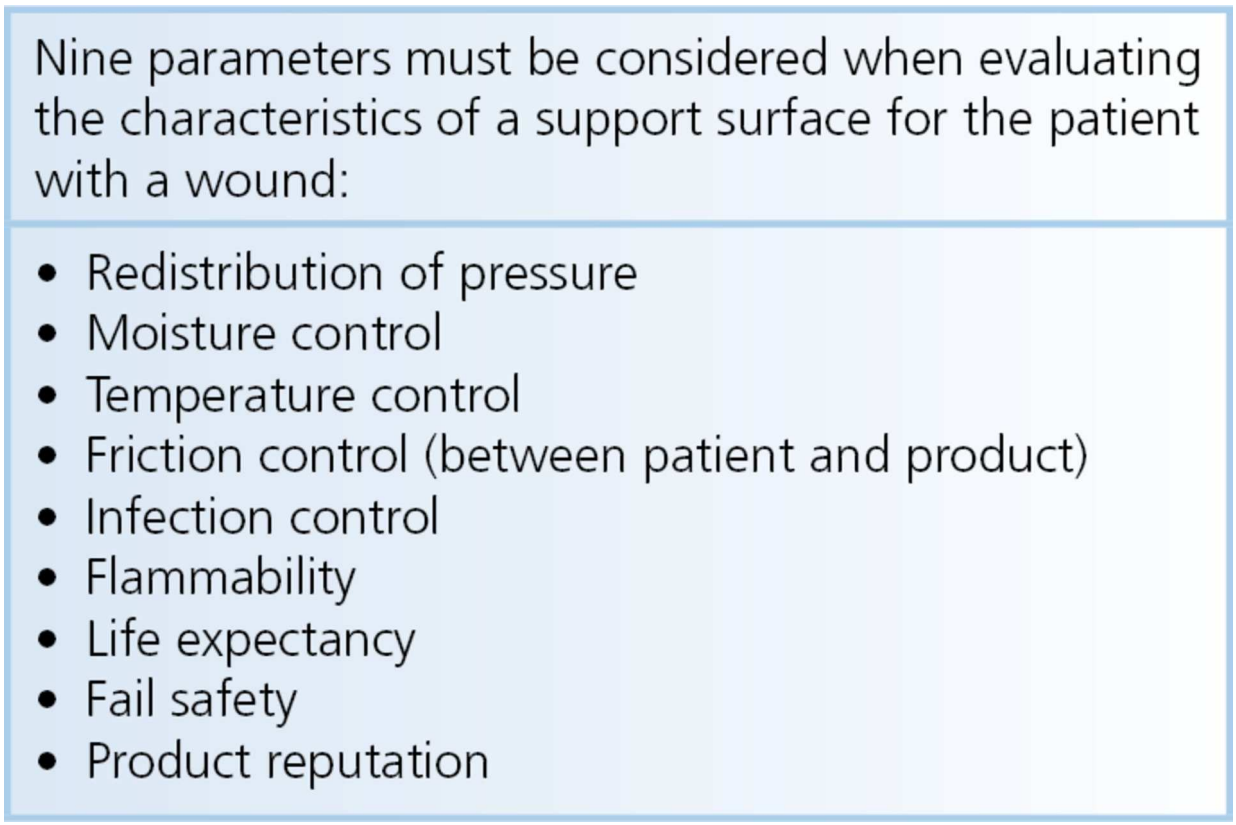
A support surface is a specialized device for pressure redistribution designed for management of tissue loads, microclimate, and other therapeutic functions. Types of surfaces include mattresses, integrated bed systems, mattress replacements, mattress overlays, and seat cushions. Unless specifically identified as a mattress or seat cushion, the term “support surface” will refer to both product categories in this chapter. Achieving a good match between the patient’s needs and the performance capabilities of the support surface has a profound, positive impact on a patient’s health and well-being; conversely, a poor match has an equally negative impact. Support surfaces redistribute the body’s weight and protect the skin’s tissue while providing for proper body alignment, comfort, and, as part of a seating system, postural control during functional movement. These effects may conflict and require clinical decision-making to strike a balance between protective and functional goals.

The need for evidence-based guidance for support surfaces to improve prevention of pressure injuries is broadly recognized in the wound care community. An ideal selection algorithm that considers an individual’s characteristics, conditions, environment, and preferences should be able to guide one to formulate a personalized recommendation. Some guidance is available for some conditions, but it’s not sufficient enough to replace good clinical decision-making and follow-up evaluations. The Wound Ostomy Continence Nursing Society developed a support surface selection algorithm for nurses, physicians, advanced practitioners (nurse practitioners and physician

assistants), occupational therapists, and physical therapists to select appropriate support surfaces for adult patients in acute, long-term acute, skilled nursing, and home care.[1] The algorithm was designed to be an evidence- and consensus-based guide for support surface selection. It was developed based on literature, expert opinion, and all published clinical practice guidelines. Preventive support surfaces are recommended for those at risk (Braden scale score ≤18).[2] The type of support surface recommended for an individual patient depends on that patient's Braden subscale scores for moisture and mobility. The Braden Scale has been validated for prediction of pressure injury risk.[2] However, in order to guide preventive interventions, clinicians look to the Braden subscales that rate specific risk factors.[3–6] Several studies have suggested that various subscale scores may be predictive of pressure injury development.[5–8] The WOCN algorithm developers focused on moisture and mobility subscales because support surface interventions, by their nature, have the most direct effects on these risk factors. Keep in mind that existing clinical recommendations need to be updated regularly to reflect new research, technology, and treatment strategies as they become available.

Knowledge of a product's composition and contents is a necessary part of the selection process. Although describing the materials and components of support surface technology may be informative, it isn't always instructive. In terms of selecting a product, the information on the function or performance of the surface is most critical, regardless of composition. In a study by Krouskop and van Rijswijk, performance parameters were emphasized when identifying nine key support surface characteristics[9] (Table 11-1).

Table 11-1 Support Surface Performance Parameters



Adapted from Krouskop, T., van Rijswijk, L. "Standardizing Performance-Based Criteria for Support Surfaces." *Ostomy/Wound Management* 41(1):34-44, January-February 1995. Used with permission.

Although function- and performance-based categorization of seat cushions and support surfaces isn't yet possible, the necessary data from clinical validation studies are being generated. Most significantly, standard tests for cushion and mattress performance have been and are being developed. These tests will provide researchers and industry with the tools needed to differentiate products based on relevant performance measures. This work is a global effort with participants from numerous countries. In the United States, the work is led by the Rehabilitation Engineering and Assistive Technology Association of North America's (RESNA's) Wheelchair and Related Seating Standards Committee.[10] The mattress standards development effort is led by the National Pressure Ulcer Advisory Panel's Support Surface Standard Initiative (S3I) Committee,[11,12] which is sanctioned by RESNA. The International Organization for Standardization (ISO)[13] coordinates and publishes both cushion and

mattress standards for worldwide use. On the clinical validation front, a study by Brienza et al.[14] has shown that support surface technology can have a positive effect on preventing pressure injuries. The best we can do now is to group the devices according to the technologies and materials used in their construction and relate the characteristics of these technologies to the factors believed to have significant effects on the prevention and healing of pressure injuries.

Soft Tissue Biomechanics

Human soft tissue consists of a variety of macrostructures, including skin, fat, muscle, vessels, nerves, ligaments, and tendons. The relative amounts and arrangement of tissue macromolecules of the skin and supporting soft tissue are adapted to their specific functions and dictate their biomechanical properties.

In most connective tissue, fibroblasts secrete the macromolecules that make up the extracellular matrix. The matrix is made up of two main classes of macromolecules:

- Polysaccharide chains of a class called glycosaminoglycans, which are usually found covalently linked to protein in the form of proteoglycans
- Fibrous proteins that are either primarily structural (e.g., collagen and elastin) or primarily adhesive (e.g., fibronectin and laminin).

Glycosaminoglycans and proteoglycans form a highly hydrated, gel-like “ground substance” in which the proteins are embedded. The ground substance is analogous to glue that fills the lattice of collagen and elastin fibers, providing lubrication and shock absorbing qualities. The polysaccharide gel resists compressive forces on the matrix, while the collagen fibers along with elastin fibers provide tensile strength and resilience.

Tissue Mechanical Properties

In general, soft tissues are anisotropic, incompressible biosolid, biofluid mixtures.[15] Obviously, soft tissue contains a substantial amount of fluid and, as a result, is largely incompressible. Moreover, tissue's response to loading has both short- and long-term phases. In the short term, tissue deforms in response to load, but the fluid within tissue also moves slowly over time from areas of greater pressure to areas of lesser pressure. This time-dependent response to loading characterizes the viscoelastic behavior of tissue. This viscoelasticity influences its mechanical properties that include stress relaxation, creep, and hysteresis.[16]

These phenomena may be graphically represented as stress–strain curves. Stress is represented as the deforming force on the y-axis, and the tissue strain (deformation) is plotted on the x-axis. When soft tissue is suddenly deformed (strained) and the strain is thereafter kept constant, the corresponding stress induced in the tissue decreases over time. This phenomenon is known as stress relaxation (Fig. 11-1). Alternatively, creep describes the progressive tissue deformation that occurs over time when stress remains constant (Fig. 11-2). During cyclic loading, such as that produced by a dynamic, or alternating pressure mattress, the stress–strain relationship demonstrated during the loading phase is different from that of the recovery, or unloaded, portion of the cycle. This effect is known as hysteresis.

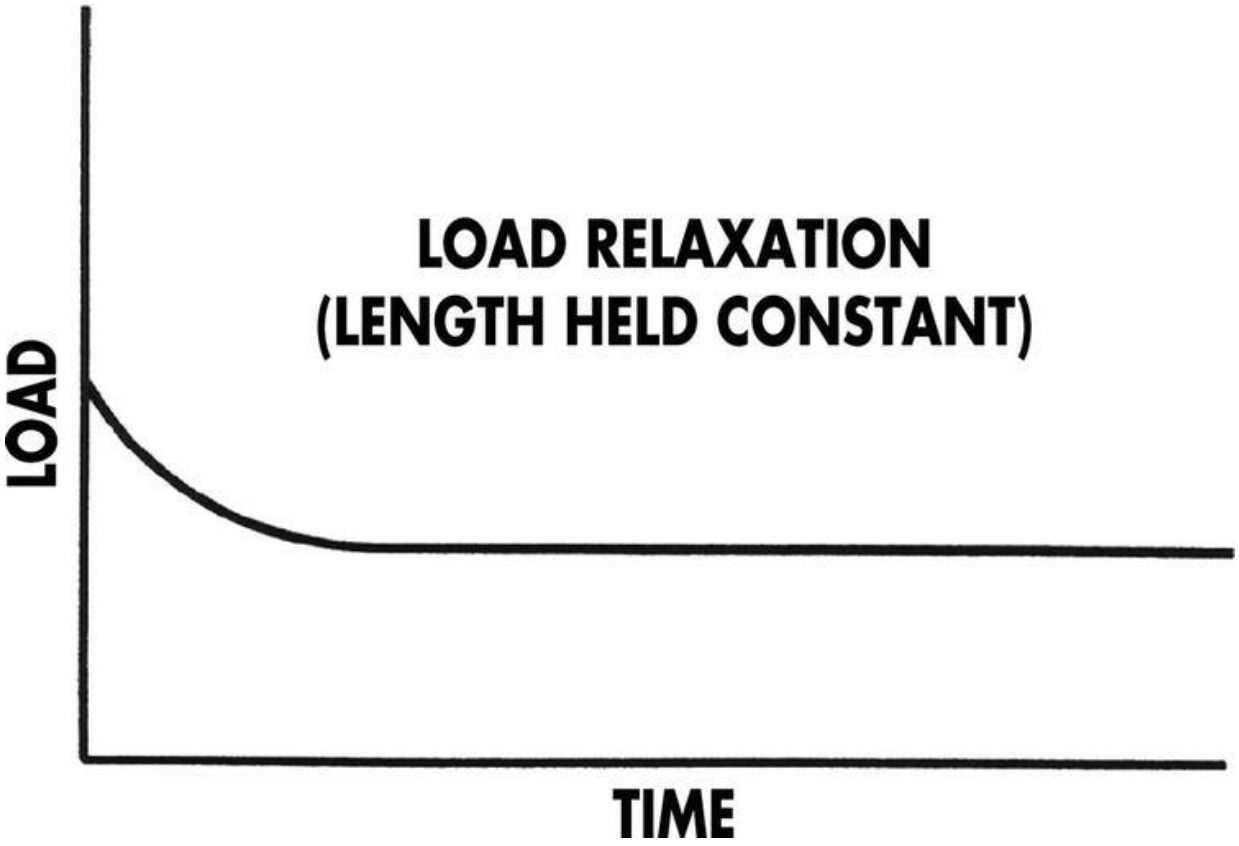


Figure 11-1. Stress relaxation phenomenon. The stress versus strain curve, shown below, illustrates the stress relaxation phenomenon. With the compression of tissue (strain) held constant, the force (stress) generated in the tissue as a result of that compression reduces over time. The degree of stress relaxation—that is, the amount of reduction in the holding force—can be determined by measuring the distance along the vertical axis between the time when the load is first applied to the time when it reaches steady state (downward sloping ends).

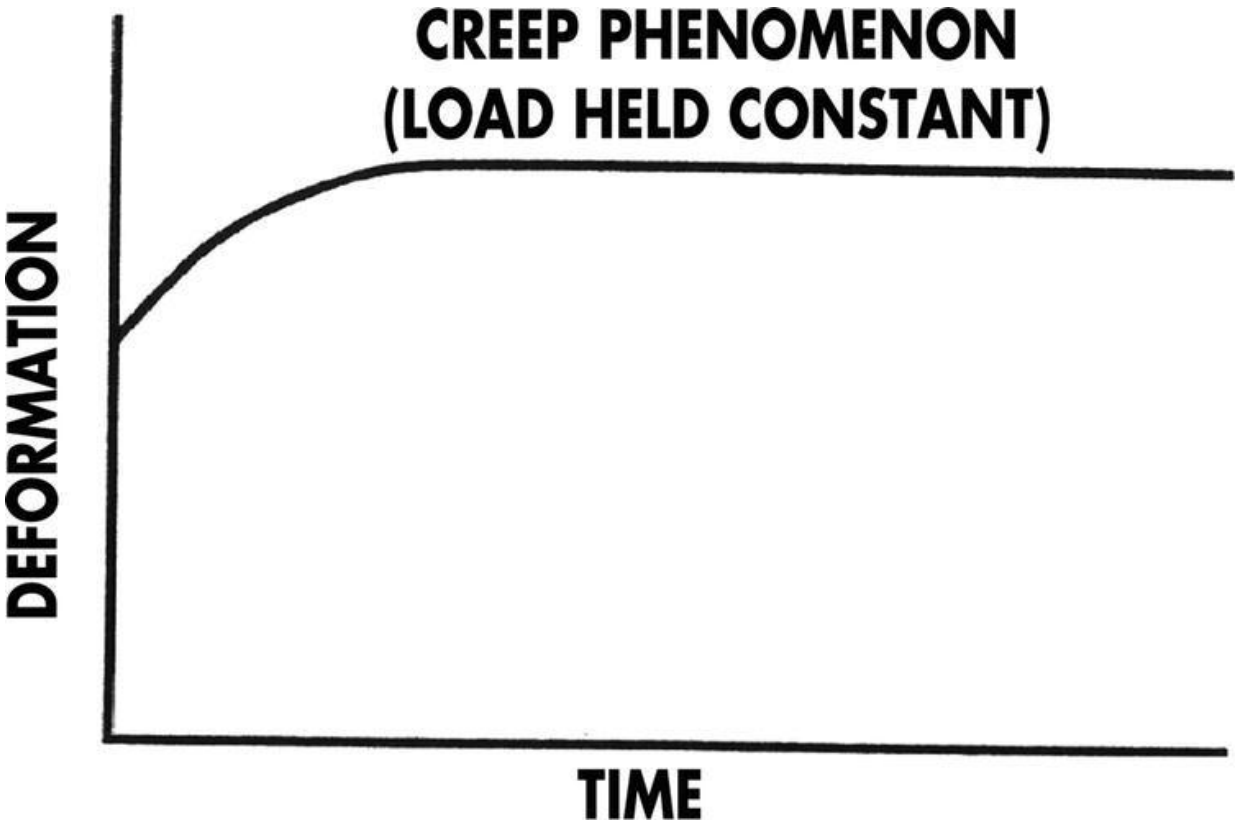


Figure 11-2. Creep phenomenon. Creep reflects the ability of tissue to resist deformation over time when the force causing the deformation remains constant. The creep phenomenon shown here indicates that the tissue progressively deforms over time without any additional force being applied. If creep were zero, the curve would be a flat line, indicating that deformation was constant over time.

Tissue Loading and Pressure Injury Formation

Body weight resting on bony prominences, such as the scapula, sacrum, greater trochanters, ischial tuberosities, and heels, can cause significant concentrations of pressure at the skin's surface and in the underlying soft tissue. The pressure peaks and the pressure gradients surrounding these peaks can put the soft tissue at risk for breakdown due to the deformation results. How tissue deformation causes pressure injuries is complex. At the cellular level, the three mechanisms most commonly cited describing how external forces result in tissue damage are (a) ischemia resulting from occluded blood vessels in the deformed tissue[17–21] (b) reperfusion injury following pressure relief and the reintroduction of oxygen-rich blood,[22,23] and (c) mechanical damage to cells caused by excessive cell structure deformation.[24–26] Certainly, combinations of these factors may be significant. A review of the evidence on pressure injury etiology to date[27,28] suggests that tissue injury due to ischemia occurs after 2 hours or more of blood flow occlusion and is caused by a cascade of events in which tissue cells exhaust their energy supply, enter a state of anaerobic metabolism that generates waste products which cannot be sufficiently cleared from the area due to impaired lymphatic flow. The accumulating waste products reduce the pH and disrupt cell function. Disruption of the sodium/potassium pump and the sodium/calcium exchange mechanisms can result concentrations that cause denaturing of proteins and DNA that leads to cell death. Deformation causes cell death more quickly. Excessive deformation (strains of 50% or more) can disrupt cell membrane diffusion properties and damage cell cytoskeleton leading to cell death in a matter of minutes.[29] Elevated skin temperature is another factor that appears to be more important than previously believed.[30–32]

Use of Interface Pressure to Assess Tissue Loading

Interface pressure is the force per unit area that acts perpendicularly between the patient's body and the support surface. Interface pressure can be measured by placing a pressure sensor between the patient's skin and the support surface. This measurement approximates the pressure on the tissue. Single sensors have been used to measure local pressure over a single bony prominence; multiple sensors integrated into a mat may be used to "map" the entire body area in contact with the support surface[18–21,26,33–36] (Fig. 11-3).

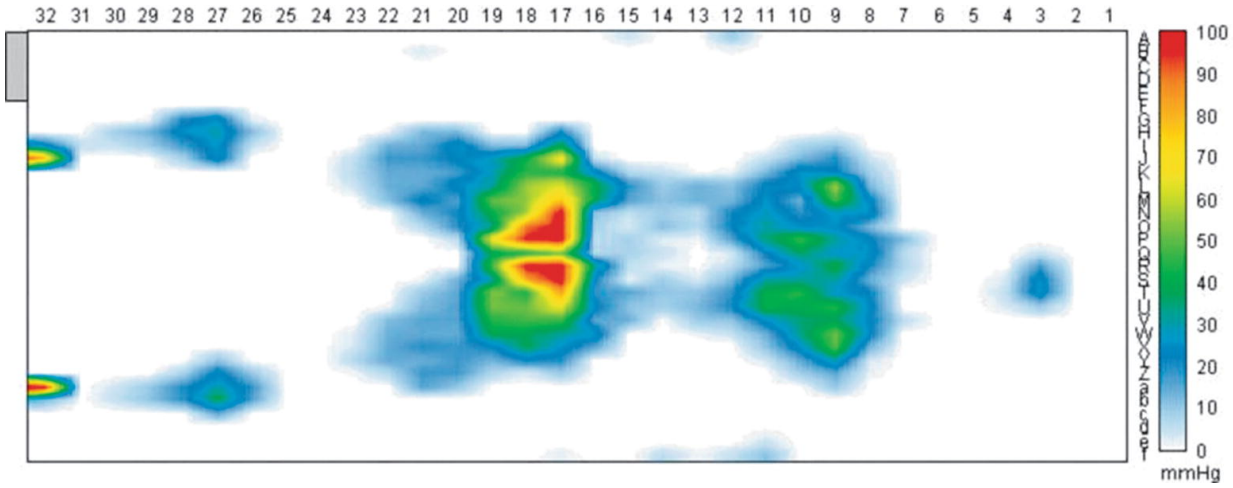


Figure 11-3. Seating, positioning, and support surfaces. Multiple sensors integrated into a mat may be used to "map" the entire body area that comes in contact with the support surface. This pressure map of a patient lying face up on a horizontal support surface shows varying degrees of pressure exerted by the patient's heels, calves, thighs, buttocks, shoulders, and head.

Interface pressure has been used extensively as a tool for predicting the clinical effectiveness of various support surfaces and for comparing products. Many research efforts have been directed toward establishing an interface pressure threshold beyond which pressure injuries would form. However, research has not identified a specific threshold at which loads can be deemed harmful across either subject populations or various tissue body sites. This is because a tissue's loading tolerance is highly individualistic and varies according to its composition, condition, location, age, hydration, and metabolic state. As a result, interface pressure should not be used as a singular indication to use a particular support surface; however, it does have clinical utility to prevent pressure injuries.[33,37,38] Measuring interface pressures for a particular person permits comparison of tissue loading across surfaces or postures. This can assist clinicians to identify unacceptable situations and help determine acceptable clinical interventions individualized to the patient or client.[39] Pressure mapping is also useful clinically to monitor changes over time when a patients' activity and posture is of interest. The main advantage of using pressure mapping technology lies in the intuitive graphical representation of how forces are being distributed on the body.

Clinical Implications of Aging

The gross morphology of the soft tissues undergoes significant changes due to aging, including decreased moisture content and decreased elasticity manifested as rough, scaly skin with increased wrinkling and laxity. Dry, inelastic skin with larger, more irregular epidermal cells leads to decreased barrier function. These changes are reflected in the tissue biomechanical properties and have been associated with increased risk of tissue injury.

At the microscopic level, flattening of the dermal–epidermal junction (rete ridges) has been observed with the height of the dermal papillae declining by 55% from the third to ninth decade of life. As the space between the well-vascularized dermis and epidermis increases, several functional changes occur. Decreases have been reported in the area available for nutrient transfer, the number of cells within the stratum basale, and the skin's resistance to shearing. A 30% to 50% decrease in epidermal turnover during the third to eighth decade of life has also been reported. This diminution in repair rate has been quantified as both decreased collagen deposition and diminished wound tensile strength. The loss of subcutaneous fat with aging decreases our protection from injury due to pressure and shearing forces between the bony prominences and the support surface. Moreover, decreased sensory perception increases the risk of injury by mechanical forces such as pressure. And, the stiffer, less elastic, drier nature of an elderly person's skin can result in tissue that tears and bleeds more easily.

Support Surface Characteristics

Prevention of pressure injuries is accomplished primarily by managing tissue loads. Support surfaces have been designed to reduce the effects of tissue loading by controlling the intensity and duration of pressure, shear, and friction. Also, attempts have been made to control the physical factors associated with increased risk through elimination of excess moisture and effective dissipation of heat.

Pressure Redistribution

Pressure redistribution is the ability of a support surface to distribute load over the contact areas of the human body. (This term replaces prior terminology of pressure reduction and pressure relief surfaces.[11] The redistribution of pressure reduces the magnitude of pressure and shear forces, both of which can cause excessive tissue distortion and damage soft tissue. Pressure (stress) is defined as force per unit area; the pressure distribution is influenced by mechanical and physical characteristics of the support surface, mechanical properties of the body's tissue, and weight distribution (posture).

Immersion

Immersion is defined as the depth of penetration into a support surface.[11] The fundamental strategy for reducing pressure near a bony prominence is to allow the prominence to be immersed into the support surface. Immersion allows the pressure concentrated beneath a specific bony prominence to be spread out over the surrounding area, including other bony prominences. For example, when a person is sitting on a relatively hard cushion, a disproportionately large portion of his or her body weight is born by the tissue beneath the ischial tuberosities. On a softer surface, the ischial tuberosities and buttocks may immerse more deeply, even to the level of the greater trochanters. With greater immersion, the body weight divided by a greater surface area results in decreased average pressure. This definition of immersion doesn't distinguish between immersion resulting from compression of the support surface and immersion resulting from the displacement of a support surface's fluid components.

The potential for immersion depends on both the force–deformation characteristics of the cushion and its physical dimensions. For fluid-filled support surfaces, immersion depends on the thickness of the surface and the flexibility of the cover. For elastic and viscoelastic support surfaces, immersion depends on their stiffness and thickness. Consider how the thickness of a seat cushion might limit the potential for immersion. If the seat cushion is 1.5

inches (3.8 cm) in depth and the vertical distance between the ischial tuberosities and greater trochanters is 2 inches (5 cm), the potential for immersion isn't enough to significantly unload the ischial tuberosities.

Envelopment

Envelopment is the ability of a support surface to conform to or mold around irregularities in the body.[11] Good envelopment implies that the surface conforms to the body without a substantial increase in pressure. Examples of irregularities are creases in clothing, bedding, or seat covers, and protrusions of bony prominences. A fluid support medium would envelop perfectly. However, surface tension plays an important role in envelopment. For example, a fluid-filled support surface such as a waterbed doesn't envelop as well as water alone. The membrane containing the water has surface tension, which has a hammocking effect on irregularities of the interface. Poorly enveloping support surfaces may cause high local peak pressures, thereby potentially increasing the risk of tissue breakdown.

Pressure Gradient

Pressure gradient, also known as *pressure differential*, is defined as the change in pressure over a distance. Although various distances have been reported in the literature, pressure gradient is expressed most commonly as a change in millimeters of mercury (mm Hg) per square centimeter or square inch. When the pressure across a surface is plotted on a graph, the slope of the curve is the pressure gradient. Because the skin and other soft tissue at risk for breakdown consist of a mixture of interstitial fluid and ground substance into which structural elements are embedded, a pressure differential between adjacent regions will result in a slowing of the flow of the tissue's fluid elements from a region of high pressure to one of lesser pressure. This flow is analogous to the movement produced when one compresses the surface of a bucket of wet sand with one's hand.

Several investigators have hypothesized that the flow of interstitial fluid caused by pressure gradients is the primary factor in the development of pressure injuries.[40,41] The flow of ground substance

and interstitial fluids from an area of high pressure is believed to increase the likelihood of intercellular contact, resulting in cellular ruptures.[40–42] This theory is consistent with the classic experimental results of several researchers showing a relationship between duration of pressure application and the magnitude of pressure that results in the formation of a pressure ulcer.[19,20,41]

Pressure gradient is intimately linked to pressure and is affected by immersion and envelopment in a similar manner. Under certain circumstances, it is possible to have pressure gradients without high pressure, and vice versa. For example, the boundary of the contact area on a support surface necessarily demonstrates a significant pressure gradient where the pressure magnitude transitions from zero outside the area of support to a nonzero value in the supported region. Despite these significant gradients, boundary areas are typically areas of lower risk for pressure ulcer development, suggesting that pressure gradient only becomes an important factor when combined with high pressure. Further research is needed to test and investigate this hypothesis.

Shear and Friction Reduction

The term shear can be used to reference shear stress or shear strain or both, and that can be a source of confusion. Shear stress refers to forces applied tangentially over an area of tissue that induce deformation (shear strain). Within tissue, stress and strain are ever-present because of the effects of gravity. Shear strain is a part of the overall tissue deformation that occurs on all supporting surfaces.[43] For example, when the head of a bed is raised or lowered, if the skin over the sacrum does not slide along the surface of the bed, or the bed does not absorb the resulting shear force by deforming in the horizontal direction, the effect will be a shearing of the soft tissue between the sacrum and the support surface. In engineering terms, the resulting shearing or deformation of the soft tissue would be referred to as “shear strain.” The characteristics of the support surface affecting this potentially harmful situation are the coefficient of friction of the surface and the ability of the surface to deform horizontally.

Friction is the resistance to motion of two surfaces in contact. Friction is a force that is dependent on the characteristics of the two interface surfaces and the contact force between them. With respect to support surfaces and tissue, friction is the force that resists sliding, and this force can induce deformation, including shear strain in the tissue.[44,45] Friction forces will induce shear strain in deep tissues even in the absence of external tangential forces. As tissue is loaded onto a horizontal surface, it tends to spread sideways. If friction prevents the skin and superficial tissues from sliding and spreading, shear stress and strain develop. Shear stress is predominant superficially where the skin is stuck to the support surface and shear strain is predominant in deeper tissues where the tissue has been displaced and spread out.

Shear strain and frictional forces can be best illustrated using the elevation of the head of the bed. When raising the head of the bed, gravity will encourage the body to slide downward to the foot of the bed. This sliding is resisted by the friction between the body and the bed linens. However, even if the skin over the sacrum does not slide

along the surface of the bed, deformation of the tissue (shear strain) can occur if the bony sacrum moves with respect to the overlying skin.

Friction cannot be fully eliminated in support surfaces. In fact, friction is needed to maintain stability whether sitting, standing, or lying. Therefore, the clinical goal is to understand friction in the context or minimizing its potential damaging influences. For example, wet skin has a higher coefficient of friction than does dry skin[44,45] and, as will be discussed below, is more susceptible to damage. Pulling someone across a bed sheet during transfer exposes the tissue to significant friction. Unweighting the person before sliding will greatly minimize the friction, because, as mentioned above, friction is dependent on the force between the contact surfaces (Fig. 11-4).

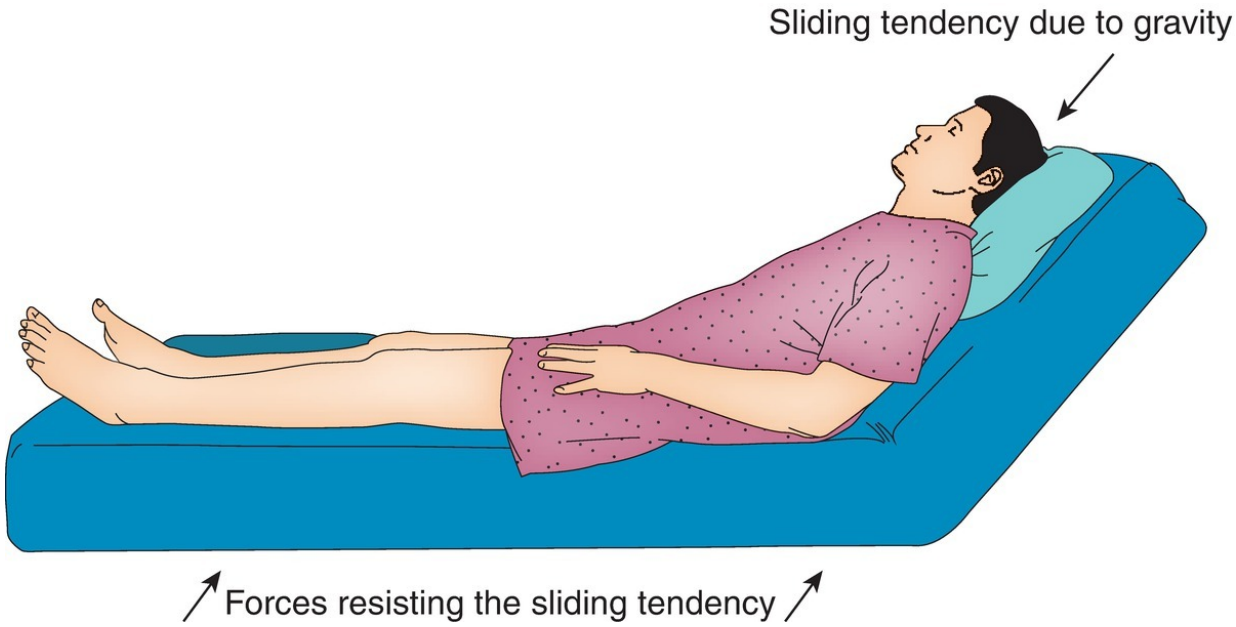


Figure 11-4. Friction and shear forces. This illustration shows the friction and shear forces acting on a person lying in bed.

Some support surface technologies protect the skin from shear better than do others. Some surfaces use a low friction interface in an attempt to minimize shear strain. Others have a low horizontal stiffness that deflects under tangential forces to minimize shear strain.

Shear strain as a contributing factor for pressure injuries is difficult to study because it is difficult to measure but has been the focus of several studies and its relative importance is becoming better understood.[46–48]

Temperature Control

One of the extrinsic factors in pressure ulcer development, temperature, has not been definitively investigated. Although hypothermia-induced vasoconstriction would seem to be related to tissue ischemia and pressure injury risk, the research evidence is not conclusive. Higher ambient temperatures have been shown to cause an increase in tissue metabolism and oxygen consumption on the order of 10% for every 1.8°F increment.[49] Thus, patients with compromised tissue already at risk for pressure injuries may have increased demands for oxygen in excess of their metabolic capabilities. Any increase in temperature in combination with pressure is believed to increase the susceptibility of the tissue to injury from either ischemia or reperfusion injury when the pressure is relieved.[50] Also, increased temperature causes an exponential increase in blood perfusion, which has been associated with an increase either in core body temperature or in local skin temperature.[50,51] For example, in one study of operatively acquired pressure injuries, the strongest predictor of pressure ulcer development was the use of a warming blanket under the patient.[52] Some clinical trials have shown that the application of repetitive surface loading alone induces an elevated skin temperature of 41°F or greater.[53] In addition, peak skin temperatures have been found to be proportional to the magnitude and duration of the applied pressure.[32,54,55] The conclusions of research vary depending upon the amount and duration of pressure that is simultaneously applied with varying temperatures.[32,54,55]

These findings clearly indicate the need for additional studies to definitively determine the effects of skin temperature modulation on the development of pressure injuries. Therefore, when choosing the right support surface for the patient, its heat transfer rate is an objective performance measurement related to its ability to control temperature effects.

Animal studies have found that increased skin temperature is related to greater tissue necrosis at all layers, and a shorter period of damaging conditions is required to cause the same amount of tissue

damage when the skin temperature is higher. Kokate[56] first investigated increased skin temperature in a swine model. They applied 100 mm Hg of pressure for 5 hours on the back of swine at four different temperatures: 25°C, 30°C, 35°C, and 45°C. Histological examination revealed that there was severe necrosis in more layers of the soft tissue with higher temperatures.

Iaizzo[57] adopted the same animal model and evaluated various combinations of skin temperature and duration of localized pressure. The histological assessment showed that temperatures as high as 45°C could cause tissue damage under localized pressure for as short as 2 hours, while sites with 25°C showed no indication of tissue damage for any duration. The protective effects of cooling the skin has also been demonstrated in humans. In particular, Tzen and colleagues[58] showed that temperature control reduces harmful effects of ischemia and demonstrated the effectiveness of local cooling for enhancing tissue ischemia tolerance in people with spinal cord injury.[59] Lachenbruch showed that temperature is a more important factor when compared to shear relative to the intensity of the reactive hyperemic response.[46] The implication of this work is that support surfaces that control the skin temperature in the range of 25°C to 27°C facilitates pressure ulcer prevention.

Moisture Control

Moisture is another key extrinsic factor in pressure ulcer development. The sources of skin moisture that may predispose the skin to breakdown include perspiration, urine, feces, and fistula or wound drainage. Excessive moisture may lead to maceration of the skin. Increases in pressure injury risk related to moisture may be due to the increase in friction that occurs between the skin and most textiles[60] or to the increase in bacterial load resulting when alkaline sources of moisture neutralize the protection provided by the normal acid mantle of the skin.

The detrimental effect of an increase in moisture adjacent to the skin has been demonstrated by tensile tests on excised skin strips in a controlled humidity environment. In a study by Wildnauer et al.,[61] the tensile strength of the strips decreased by 75% with an increase in relative humidity from 10% to 98%. Skin with such reduced strength may be more prone to mechanical damage from shear stress and could easily be abraded.

Materials, Components, and Features Used in Support Surface Systems

The components and materials described here are the most commonly used in support surface systems and may be used alone or in combination. The most basic support systems use foam with a protective cover. More specialized support systems, low-air-loss systems for example, necessitate a specialized cover and make use of an air pump. A support surface system is a general term for any specialized device for pressure redistribution designed for management of tissue loads, microclimate, and/or other therapeutic functions.[12] A cushion, for example, is a support surface for sitting while a mattress is a support surface for lying down.

Support surface systems are composites of diverse materials. Some are more familiar, such as a layering of different foams with a breathable cover, but others are more specialized, such as silicon beads encased in a semipermeable sheeting with pressurized air forced through them so that they take on the characteristics of fluid. The performance of any support surface system is dependent on all elements combined; no one element attends to all needs of a user. When any element is compromised or altered from suggested specifications, whether a cover or interior components, the entire system risks becoming compromised.

In order to achieve the desired outcome for the user, each support system needs to be selected for that user while considering both purpose and care environment. Such factors that impact support surface systems include:

1. The heat of a body lying on them
2. The moisture from sweat that permeates into them
3. The moisture and organic load from incontinence that is deposited on them
4. The compressive load, the size of the occupant
5. The expected life
6. Housekeeping and care practices

    a. Frequency of bedding changes
    b. Cleaning, disinfection, and rinsing practices
7. Therapies or procedures carried out on the surface
    a. Physical therapy
    b. Dressings, wraps, and device changes
8. Transfer devices
    a. Transfer boards
    b. Slings and sheets and hoists
9. User sliding on the surface versus ploughing through the bulk of the surface; that is, sliding on top, or sinking in and still sliding.

The clinical setting in which a support surface system is used is also a critical factor. A support surface used in a walk-in clinic has radically different requirements than a support surface used in an intensive care unit of a hospital. When selecting a support surface system, clinicians should be aware that “there is no international definition of what constitutes a standard foam hospital mattress, and, indeed, this changes over time within countries, and even within hospitals”.[62,63]

Support Surface Materials

The internal elements of a mattress/cushion support surface may be comprised of foam, gel and gel pads, fluid-filled bladders, viscous fluid, air, or combinations of these.

Polyurethane Foam

Polyurethane foam (PUF) may be elastic or viscoelastic.[20] Elastic foam is a type of porous polymer material that conforms in proportion to the applied load.[64] Consequently, greater loads result in predictably greater deformations, and vice versa. If time is a factor in the load versus deformation characteristic, the response is considered to be viscoelastic, which is discussed separately. The response of support surfaces made from high resilient foam (HR Foam) is predominately elastic. Elastic foam is resilient, meaning that it returns to its nominal shape or thickness once load is removed. As with all materials, there is an “elastic limit” to the material’s ability to respond to bearing the load of a body.[65]

All foams experience a life cycle composed of break in, highly resilient elastic performance, and the decay period when sag or stiffening occur, also known as foam fatigue. One indication of foam fatigue is an inability of foam to return to its original unloaded condition. PUF is comprised of a polymer matrix with entrained and distributed gas bubbles creating open or closed cells. Open-cell foam is a permeable structure in which the cells are interconnected which allows air and/or fluids to “communicate” or pass between the cells. Closed-cell foam is a nonpermeable structure comprised of isolated cells that are not connected. As a result, closed-cell foam tends to be stiffer than open-cell foam simply because it does not compress as readily since air and/or fluid is trapped and cannot move.

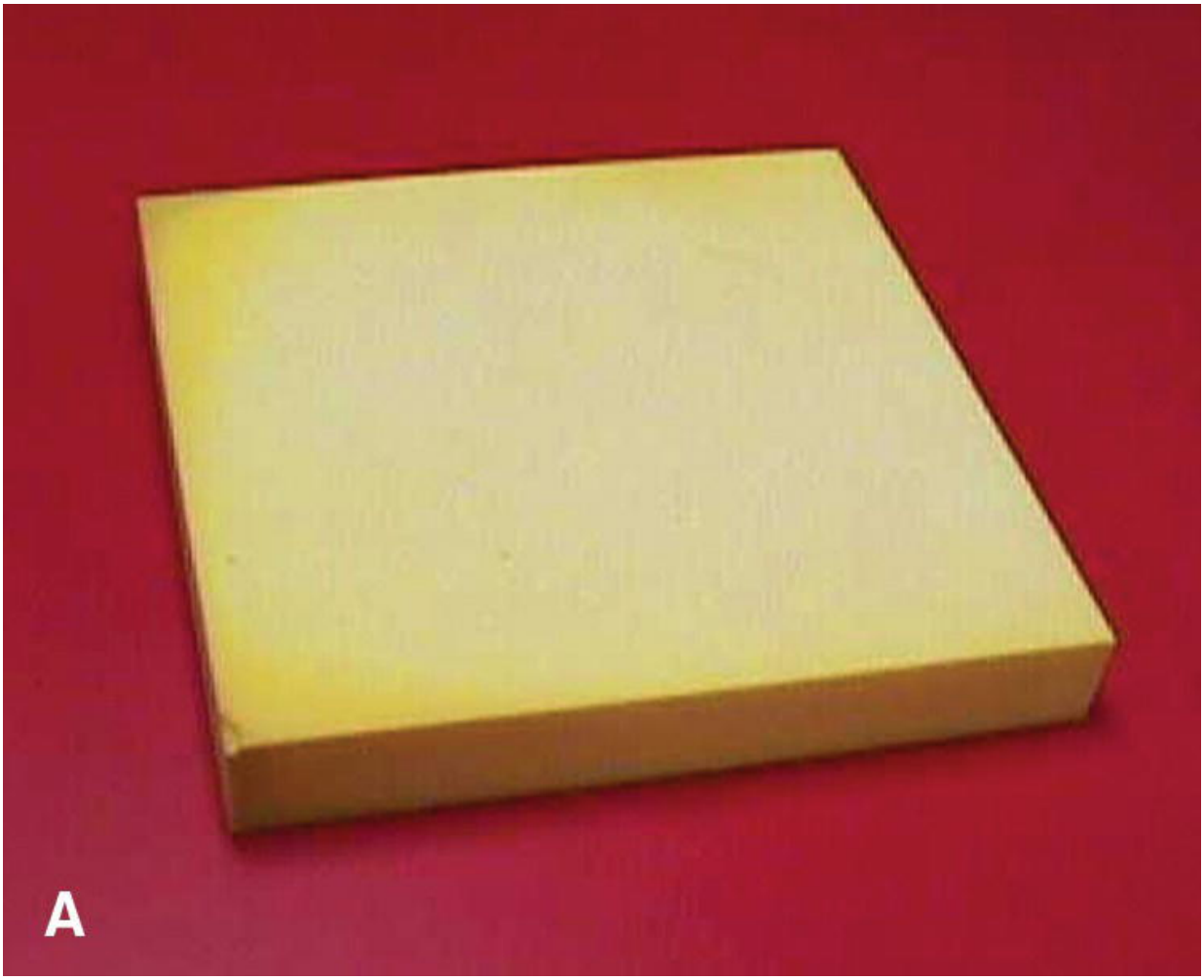
PUF products are characterized by density and by stiffness. Density reflects the weight of the polymer in the matrix of the foam and stiffness reflects the force required to deflect the foam a given amount. Stiffness is governed by the elasticity of the matrix and the amount of chemical crosslink bonds present in the polymer. High

crosslinking generates lower elastic elongation, and low crosslinking creates high elastic elongation in the polymer matrix. Both stiffness and thickness impact PUF's ability to immerse and envelop; soft foams permit greater immersion and envelopment compared to stiffer foams, but thicker foams have a lower risk of losing the ability to compress further with the addition of more load, also known as "bottoming out."

When PUF is made softer, fewer materials are required which makes the foam less expensive but also less durable. Inversely, making foam heavier requires more materials, making the foam more expensive but also more durable. Optimum performance can be achieved through a careful combination of heavy and soft, balancing longevity against cost while considering the needs of the end-user.

Many PUF products consist of layers of foam of varying stiffness. This laminar design is used to optimize the performance of the end product by combining the different strengths of the several foam types and materials. For instance, in a laminar foam product, the softer foam is on top, thereby allowing immersion and envelopment of the body while denser foams comprise the bottom layer to help a support system manage the load placed upon it by a user and avoid "bottoming out."

PUF used in seat cushions can also be contoured that increases the contact area and immersion, thereby reducing average pressure and pressure peak[65] (Fig. 11-5). PUF is also used in combination with other materials such as air and fluid. A foam base offers postural stability while the elastomer, air, or fluid provides an enveloping interface. An ideal combination of characteristics for an elastic support surface is resistance that adjusts to the magnitude of compressive forces.[9,66] The support surface should be soft and thick enough to provide good immersion and envelopment but have high enough compression resistance and thickness to fully support the load without approaching maximum compression.


A

B

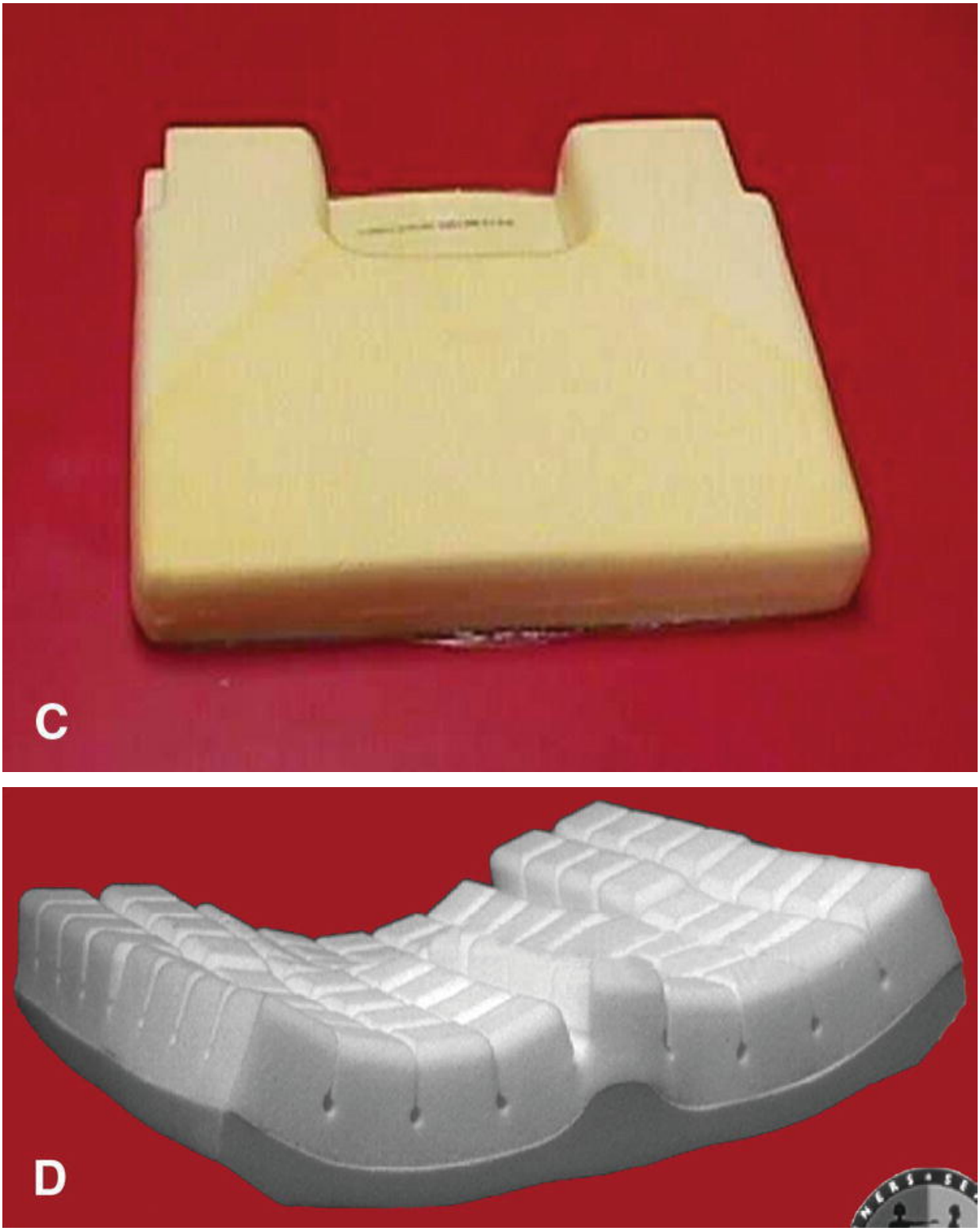


Figure 11-5. Elastic foam seat cushions. These photos show four different types of elastic foam seat

cushions: **(A)** Flat. **(B)** Contoured. **(C)** Cut-out. **(D)** Segmented.

Near maximum compression, sometimes referred to as “bottoming out,” the foam becomes much more rigid and provides too high a reactive force and too great a concentration of pressure the tissue damage threshold. Bottoming out does not necessarily mean that a patient has penetrated completely through the foam to press down on the frame or structure supporting the foam. This might be envisioned as the foam losing the ability to compress further with the addition of more load. Over time and with extended use, foam degrades and loses its resilience. This decreased ability can result in higher interface pressures if it causes bottoming out. Krouskop estimated that in approximately 3 years, a foam mattress wears out.[9]

PUF tends to increase skin temperature because its materials and the air they entrap are good insulators, much like putting on an extra coat. User movement can also increase heat transfer rates. Mean temperature increases of 6.1°F (3.4°C) and a 10.4% increase in moisture at the skin surface have been recorded on foam products after 1 hour of contact.[67]

Viscoelastic Foam (Memory Foam)

Viscoelastic foam is a special type of open cell foam that compresses under load like regular elastic PUF but has other unique qualities. Viscoelastic foam is sometimes referred to as “memory foam” because it contours to the body slowly and its elastic response causes it to return to its original shape an extended period of time. This is a characteristic of stress relaxation and allows viscoelastic foam to maintain its deformed shape for a few moments before returning to its original unloaded shape (ergo, “memory foam”).

Viscoelastic foam comes in a variety of stiffness so it can allow immersion similar to a comparable elastic PUF. The difference lies in its enveloping quality. Because of the stress relaxation, viscoelastic foam envelops better than elastic foam. However, viscoelastic foam also exhibits creep, meaning that, over time, it will continue to deform

under the same load, and after many loading cycles, it will fail to return to its original shape.

Viscoelastic foam products are temperature sensitive and become softer at temperatures near that of the human body. Mean temperature increases of 5°F have been reported for viscoelastic foam.[67] This characteristic allows the body to immerse more into the foam and can improve contact area and lower interface pressure. Because both creep and temperature sensitivity can lead to a greater potential of bottoming out, viscoelastic foam, like PUF, must be chosen according to the specific needs of the patient for both seating and mattress applications (Fig. 11-6).

Figure 11-6. Viscoelastic gel seat cushion.

Reticulated Foam

Reticulated foam is a specialized type of open cell foam where gas or air has been replaced with a combustible mixture of gasses and ignited. The result is a low-density, web-like structure with a porosity typically at 95%. Reticulated foam trades a decrease in resistance to compression for an increase in tensile properties, such as high resistance to tearing and elongation and very high air flow or exchange.

Gels

Soft elastomers are rubber-like materials and are sometimes referred to as solid gels. For use in support surfaces, gels are often used on top of other materials because they tend to be heavy yet have desirable body contact properties. Gels have a high heat capacity and conductivity. This is evidenced by the many elastomeric products designed to be heated or frozen to provide relief to a sore body part. When used as a support surface, an elastomer will absorb body heat so the interface temperature will not rise as quickly.[9] This allows elastomer gels to feel cooler once they come in contact with the body. Overtime, the elastomer will eventually reach heat capacity and the interface temperature will rise unless the body is repositioned—via pressure reliefs or other repositioning—to allow the heat to dissipate. Depending on the elastomer and cushion cover, moisture at the interface can also rise.[66] Repositioning on a recurring schedule is advised with these heat absorbing products to allow the trapped heat to escape rather than hold it against the skin.

Fluid-Filled Bladders and Compartments

Fluid-filled products may consist of small or large chambers filled with air, water, or other viscous fluid materials, such as silicon elastomer, silicon, or polyvinyl. The fluid flows from chamber to chamber or within a single chamber in response to movement and requires no supplemental power. The term “air-flotation” is sometimes used to describe interconnected multichamber surfaces (Fig. 11-7).

Figure 11-7. Fluid-filled products. As the photos demonstrate, fluid-filled products come in a variety of

forms: **(A)** Cushion. **(B)** Mattress.

With unpowered air-filled products, be careful to maintain the correct levels of inflation in air cushions to achieve optimal pressure redistribution. Under-inflation allows bottoming out, and over-inflation increases load on the tissue by reducing the area that the body weight is distributed over. For surfaces with viscous fluid bladders, such as seat cushions, it is important to monitor the distribution of viscous fluid and manually move it back to the areas under bony prominences if it has moved away from these areas. This is also true of positioners that have no shape memory.

Most fluid-filled products permit a high degree of immersion, allowing the body to sink into the surface. The surface conforms to bony prominences, effectively increasing the surface pressure distribution area and lowering the interface pressure by transferring the pressure to adjacent areas. These products are capable of achieving small-to-modest deformations without large, shape-restoring shear forces. In a direct comparison of interface pressures with air-fluidized and low-air-loss beds, zero memory fluid mattress was shown to relieve pressure as effectively as the air-fluidized and low-air-loss surfaces used in the study.[68]

Skin temperature is affected by the heat capacity and thermal conductivity of the fluid material contained in the support device. Air has a low heat capacity, whereas water has a high heat capacity. The oil-based viscous material used in zero memory products also has a high heat capacity, and skin interface temperature will not rise as rapidly compared to PUF support surface systems.[68]

Given the large variety of materials used in fluid bladders, it is difficult to generalize on the moisture control characteristics of these products. However, the insulating effects of rubber and plastic used in some fluid-filled products have been shown to increase the relative humidity due to moisture transpiration and perspiration.[67]

Air

Air-based support surface systems include air-fluidized, low-air-loss, alternating pressure, and lateral rotation products. The features of these support systems can be used alone or in combination with features of gels, PU foams, and other cushioning elastomers.

Air-Fluidized

A support surface with an air-fluidized feature provides pressure redistribution via a fluid-like medium created by forcing air through beads as characterized by immersion and envelopment.[62] These support surface systems were originally developed in the late 1960s for use with burn patients but are now also used to provide maximum pressure redistribution for free-muscle flaps, complex Stage 4 Pressure Injuries, Deep Tissue Pressure Injuries, or pain control in bony metastasis. The constant high airflow can also be used in cases of excessive tissue maceration. These support systems contain granular materials such as silicon beads encased in a breathable polyester sheeting. The granular material takes on the characteristics of a fluid when pressurized air is forced up through them. In some models, the fluidization feature is variable, permitting individualization based on the user's needs. While the fluidization feature should be active almost constantly, it can be temporarily turned off for transfers in and out of bed, and for positioning for wound care. Some models of air-fluidized beds allow adjustment of the head-of-bed section and consist of a low-air loss surface. The two different surfaces used in the same adjustable bed frame provide optimum pressure redistribution and caregiving flexibility.

These products aid in the reduction of evaporative water loss and may need additional monitoring to ensure that excess moisture is not removed from the occupant (Fig. 11-8). Incontinence and other body fluids flow freely through the sheet. To prevent bacteriologic contamination, the bed must be pressurized at all times, and the sheet must be properly disinfected after each occupant and at least once per week with long-term use by a single occupant.[62,69]

A

Figure 11-8. Air-fluidized **(A)** and low-air-loss **(B)** beds.

Air-fluidized support surface systems use fluid technology to redistribute forces on the body through the principle of immersion while simultaneously increasing envelopment, reducing both loading forces and shear forces on the skin. These products permit the highest degree of immersion currently available among support surfaces. The surface of air-fluidized systems conforms to bony prominences by permitting deep immersion into the surface—almost two-thirds of the body may be immersed.[70] The immersion effectively lowers the interface pressure by increasing the area that forces are distributed over. The greater deformations possible with this technology enable the transfer of pressure to adjacent body areas and away from the at-risk bony prominences. Envelopment and shear force are minimized.

The pressurized air in these support systems is generally warmed to a temperature level of 82.4°F to 95°F (28°C to 35°C); however,

warming may be beneficial or harmful depending on a patient's needs. For example, heat may be harmful to patients with multiple sclerosis, but beneficial for patients in pain. The beneficial effects must be balanced against the increasing metabolic demands of the tissue. Air-fluidized beds are advantageous for burn patients due to their effectiveness in managing body fluids.

Low-Air-Loss

Low-air-loss is a feature of a support surface that provides a flow of air to assist in managing the heat and humidity (microclimate) of the skin.[71] Low-air-loss systems use a series of connected, air-filled cushions or compartments, which are inflated to specific pressures to provide loading resistance based on the patient's height, weight, and distribution of body weight. An air pump circulates a continuous flow of air through the device, replacing air lost through the surface's pores. The inflation pressures of the cushions vary with the patient's weight distribution. Some systems have individually adjustable sections for the head, trunk, pelvic, or foot areas. As with other fluid-filled surfaces, the temperature of the skin is affected by the specific heat of the fluid material. However, the constant air circulation and evaporation tend to keep the skin from overheating.

In low-air-loss systems, the patient lies on a loose-fitting, waterproof cover placed over the support surface. The waterproof covers are designed to let air pass through the pores of the fabric and are usually made of a special nylon or polytetrafluoroethylene fabric with high moisture–vapor permeability. Manufacturers have addressed the problem of skin dehydration by altering the number, size, and configuration of the pores in the covers.[59,70] The material is very smooth, with a low coefficient of friction; in addition, it is impermeable to bacteria and easy to clean.[70] Low-air-loss devices have been shown to prevent buildup of moisture and subsequent skin maceration.[72]

Alternating pressure is a feature of a support surface system that provides pressure redistribution via cyclic changes in loading and unloading as characterized by frequency, duration, amplitude, and rate of change parameters over the "active area" of the surface.[71]

These systems contain air-filled chambers or cylinders arranged lengthwise or in various other patterns. Air or fluid is pumped into the chambers at periodic intervals to inflate and deflate adjacent chambers in opposite phases, thereby alternating the force applied to the tissue from one area to another in cyclic fashion. The frequency of the alternating pressure feature can have an effect on its use. For example, very short peak inflation, and the cycling pattern from distal to proximal over time appear to have a dramatic effect on increasing lymphatic flow.[73] Low-air-loss systems show greater benefit on lower body mass index by increasing sacral skin blood flow.[74]

Rather than increasing the surface area for distribution through immersion and envelopment, alternating-pressure devices distribute the pressure by shifting the body weight to a different surface contact area. This may increase the forces applied to the tissue of that area during the inflation phase and may also induce a resulting higher tissue distortion. The effectiveness of these devices relies on the cyclic nature of the loading and the fact that short loading periods are tolerated by the body.[75]

Alternating pressure technology has the same potential as any other fluid-filled support surface to influence temperature at the interface, and therefore, care must be taken to maintain the correct levels of inflation. The skin moisture control and temperature control characteristics of alternating-pressure surfaces also depend on the characteristics of the cover and supporting material (Fig. 11-9).

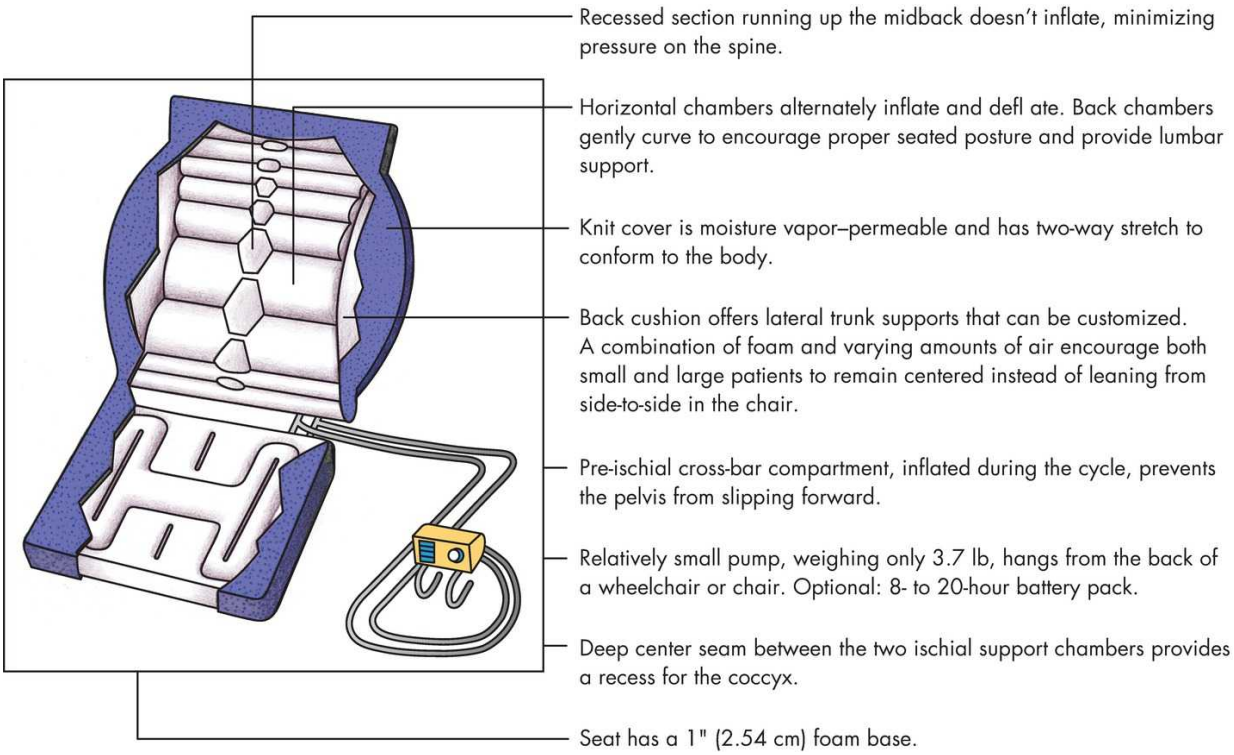


Figure 11-9. Alternating-pressure integrated cushions. The illustration shows the characteristics of alternating-pressure integrated cushions.

Lateral Rotation

Lateral rotation is a feature of a support surface that moves the patient in a regular pattern around a longitudinal axis as characterized by degree of patient turn, duration, and frequency.[76] Although these devices have been used for several decades for other medical purposes, such as pulmonary therapy and spinal alignment, research is conflicting on their use for pressure ulcer treatment. No matter what the therapeutic goal for use of a lateral rotation support surface, routine assessment of the skin is essential for early identification of pressure/shear injury. This assessment can be challenging but is possible through the use of "hatches" on the undersurface of the bed. In addition, a more recently developed lateral rotation device has the ability to laterally rotate a patient while prone. (Fig. 11-10) Lateral rotation may be continuous (i.e., on an automatic timed cycle) or

manual, where the bed is rotated and locked in that position.[77] While rotating, the body is held in position (“packed”) by sequentially positioned bolsters that maintain neutral spinal alignment. The therapy works by positioning the patient in such a way so that one lung is higher than the other to prevent pneumonia.

Figure 11-10. Lateral rotation bed which allows prone positioning; lateral rotation bed.

This therapy is not intended for patients with cervical or spinal fracture, intracranial pressure instability, or long bone fractures. There are reported cases of skin shearing that occurs from the constant sliding from one side to the other. Thus, lateral rotation must be used judiciously and cautiously. It is also used in assisting care staff in turning a patient. This is later rotation for turn assist, and it is able to begin the process of turning the occupant on their side and reduce the work required by the care staff.

According to the NPIAP “Whenever lateral rotation features are used, the risk for shear injury exists. Shear force tangentially strains the skin (through stretching) and interrupts blood flow of the skin. Unless the individual is properly positioned and bolstered, shearing can occur with every rotation, causing a new ulcer or worsening existing ulcers”[77] (Table 11-2). Secure positioning to reduce shear may be challenging where devices such as chest tubes may preclude snug packing. While packing a patient on the surface may reduce shear forces, off-loading of bony prominences such as occiput, heels, and elbows is still necessary. It should also be noted that the new ISO 20342 Standard requirements for support surfaces requires that substantial validation of safety be performed before these designs are put in use due to the risk of entrapment at one of the bed rails during a patient’s migration toward the bed rail in the cyclic movement process.

Table 11-2 Recommendations for Positioning and Repositioning

Recommendation	Strength of Evidence	Strength of Recommendation
Reposition all individuals at risk or with pressure injuries on an as needed basis if not contraindicated schedule	B1	↑↑ Definitely do it Strong positive recommendation, Definitely do it
Determine frequency of repositioning taking activity, mobility, and ability to independently reposition into account	B2	↑↑ Strong positive recommendation Definitely do it
Implement reminder schemes to improve repositioning schedule adherence	B1	↑ Weak positive recommendation, Probably do it
Consider using continuous pressure mapping while in bed	C	↔ No specific recommendation
Reposition the individual using manual methods or equipment that minimize shear force and shear strain during the maneuver	B2	↑ Weak positive recommendation Probably do it
Use the 30° side lying position versus the 90° position	C	↑ Weak positive recommendation Probably do it
Initiate frequent small body position shifts for individuals too unstable for regular repositioning and to supplement regular repositioning	C	↑ Weak positive recommendation Probably do it
Keep the head of bed as flat as possible	B1	↔ No specific recommendation
If possible, avoid extended use of prone positioning	B1	↔ No specific recommendation
Promote sitting out of the bed versus in the bed	B1	↑ Weak positive recommendation Probably do it
Implement early mobilization program that increases activity and mobility as soon as possible	C	↑ Weak positive recommendation Probably do it

Adapted from European Pressure Ulcer Advisory Panel, National Pressure Injury Advisory Panel, and Pan Pacific Pressure Injury Alliance. In: E. Haesler, ed. *Prevention and Treatment of Pressure Ulcers/Injuries: Clinical Practice Guideline. The International Guideline*. EPUAP/NPIAP/PPPIA, 2019. Additional recommendations are available in the guidelines.[77]

Covers

A support system cover provides protection for internal elements by presenting a barrier against the passage of contaminants, such as moisture, body fluids, and microorganisms while ideally keeping a patient comfortable through regulating moisture and temperature. Although the cover is one part of the support system, it should be considered an active element within a greater whole. Support systems need to have a top surface that is a complete barrier. To aid in this a hydrophilic, rather than a microporous surface transfers water in a state of vapor only, keeping the underlying components free of contaminants and microorganisms. Older support system cover materials were comprised of vinyl with a viscous waterproofing solution coated straight onto the textile. Modern covers come in a wide array of polyurethane transfer coatings on a synthetic textile substrate. Polyurethanes (PU) are a group of polymers with thousands of different types commercially available that vary in performance including moisture vapor permeability, chemical resistance, and resistance to stretch. An ideal PU coating would have high breathability and high resistance to chemicals, such as those found in cleansing agents, but these qualities are mutually exclusive. Current support systems negotiable a balance between them. A cover should never be punctured for any reason. For example, while poking holes in a low-air-loss mattress to allow air to blow on a patience may sound like a positive step in patient care and comfort, the holes become a channel for incontinence and other body fluids. Such actions inevitably lead to contamination and eventual compromise of the internal elements of the support system.

Stretch

Stretch indicates a material's ability to be pulled without breaking the fibers while retaining the ability to return to its original length. The ability of a cover to stretch offers greater freedom of movement to underlying materials of a support system while resisting abrasion. It also serves to enhance a user's comfort. Stretch can be 2-way or 4-way, but descriptions of these vary country to country. In the United

States, a 2-way stretch fabric can be pulled lengthwise, that is, a 1-foot section stretching to 3 feet. In Europe, this is simply called stretch fabric. In the United States, a 4-way stretch is a 1-foot section stretching lengthwise while also stretching top to bottom. In Europe, this is called a 2-way stretch. While rarely specified in mattress covers, modulus of the stretch is also a factor. The modulus is a measure of resistance to being stretched. If a fabric is easy to stretch, it has a low modulus. If it is hard to stretch, it has a high modulus. But the degree of stretch is also important. A fabric that is easy to stretch out 50% more in length is different than a fabric that has a hard stretch to 100%. The different types of modulus of fabric is most easily recognizable in undergarments such as those made from spandex that are designed to be elastic with varying degrees of hard or soft stretch depending on the utility of the garment, that is, compression undergarments for sports and work-outs versus daily wear for general support and comfort.

Coatings

PU coatings allow materials beneath them to stretch while introducing a moisture-vapor permeability (MVP) factor unavailable when using direct coated vinyl. Covers may be coated with or contain antimicrobial solutions.

Maintenance and Cleaning

Maintenance of support surface systems is of the utmost importance. Mattress covers can become damaged from sharp objects, needle sticks, and strikethroughs. Abrasions occur from handling, regular wear and tear of use or moving. When a PU coating breathes, it swells, making it more vulnerable to abrasion. Incorrect observance of cleaning and decontamination protocols can damage or degrade a cover's protective coating, causing possible delamination and making it more susceptible to an increased risk of compromising not only the protective cover but also the internal components. Contamination of internal components can lead to the entire support system's failure. Even if some components can be repaired, such as a patch over a puncture in the cover, or replaced, such as zipping on an entirely new cover, there is no guarantee that the support system as a whole is uncompromised.

Cleaning Agents

Because support systems can become a hotbed for germs and infection, cleaning agents are invaluable in the care and disinfection of support system covers, especially in the prevention of cross infection. These agents can often, though unadvisedly, default to a bleach product or hydrogen peroxide with aggressive elements, especially in facilities responsible for care and maintenance of a large number of support systems where efficiency of readying a bed for the next patient outweighs considerations of the effects of the cleaning agent on that bed. Aggressive cleaning that does not consider manufacturer's specifications can compromise the covers and internals, intensifying the problem of contamination to the point that the entire support system may need to be disposed of. Most mattress covers have a polyester polyurethane top coat. Polyurethane coatings offer a wide range of performance with varied levels of moisture vapor permeability (MVP) and chemical resistance. Some have a polycarbonate backbone that gives them a higher resistance to chlorine-based cleansers.

While PU coatings are most resistant to chlorine bleach, they are not immune. Exposure time to chlorine is the important factor, not the number of wipes. If a surface is not properly washed after each application of a cleaning agent, that agent can build up and even be absorbed into the bed cover or internal components, aggravating a user's existing health conditions or developing new ones such as skin dermatitis. Even in the presence of appropriate cleaning agents in the appropriate concentrations, polyurethane coatings swell, making them more prone to damage during regular use that can lead to an increased risk of contamination of the underlying support material. Incorrect cleaning, such as with a higher concentration or incorrect chemical agents, can directly damage and degrade the coating, even to the point of delamination. Some products can even deactivate disinfectant, and new products remain antimicrobial without damaging the cover. PU polymers with greater chemical resistance are available, and recent tests have shown improved performance when measured against standard coatings, but they drive up cost. While promising, chemical resistance of coatings varies considerably manufacturer to manufacturer. Testing of performance is ongoing, and development of consistent standards is underway.

Sporicidal Products

In response to superbugs such as MRSA and *Clostridium difficile* (C. diff), new antimicrobial and sporicidal products have been developed, many of them pH neutral. But claims of sporicidal effectiveness are most often based on cleaning hard surfaces rather than PU coatings of stretchy materials. Not much testing has been done. Bleach may seem like an obvious and simple solution that will easily eliminate infectious elements, but the use of bleach is like melting a marshmallow with a blow torch. In the short term, contaminants are removed simply and effectively, but only on the surface. In the long term, the cover becomes increasingly susceptible to all types of damage from normal use, risking the compromise of internal elements as well as patient health if in the presence of a buildup cleaning agents. Bleach may cause as many problems as it solves. Manufacturer recommendations should be consulted for maintaining covers of any support surface system.

Manufacturer Recommendations

Manufacturers suggest that surfaces should be cleaned before being decontaminated/sterilized with strength-appropriate cleaning agents. Surfaces should be rinsed after cleaning/decontamination to prevent build-up of any residual chemicals. As a last step, the surface should be thoroughly dried before use or being placed into storage which will help reduce swelling in the polyurethane coating and decrease its susceptibility to physical damage (Table 11-2).

Matching Support to Patient Needs

Although widely used, support surfaces have neither performance standards nor criteria for function that can be tested against clinical outcomes. Indeed, the basis for effective function is not known or is poorly understood, for such common products as wheelchair seat cushions and horizontal support surfaces intended for skin protection and healing of wounds. Despite this, clinicians must have some basis for decision-making regarding the selection of these products. In 2015, the Wound Ostomy Continence Nursing Society developed an Evidence-and-Consensus-Based Support Surface Algorithm that can be used by bedside clinicians and tailored to an institution's approved catalog of support surfaces.[1] The following key questions should be used to guide the decision-making process.

What Are the Patient's Specific Load Management Needs?

Regardless of body position, the first step in determining an individualized protective intervention is to perform a general physical assessment and functional evaluation. Much of this information will then be used to assess the patient's risk for pressure ulcer development.

General Physical Examination and Functional Evaluation

Patient evaluation is described elsewhere in this text; however, several additional items are germane to the selection of a support surface. These include assessing the capability of the patient for specific bed mobility (movement on the surface, ingress and egress, and ability to place supportive devices), the number of available turning surfaces, the time spent in lying in bed or sitting per day, the number of devices or pillows needed for positioning, the patient's body weight and its distribution, and the presence of contractures.

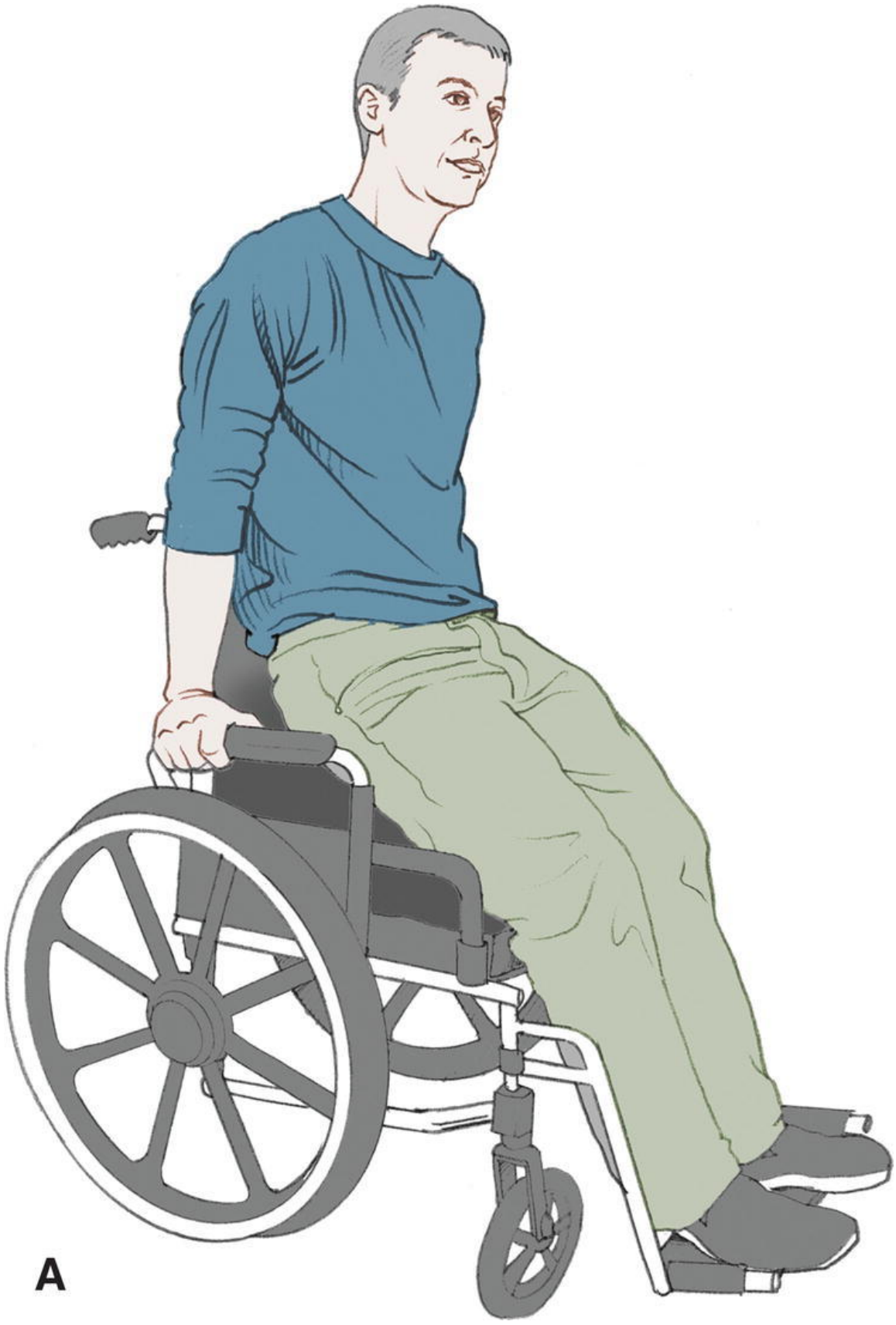
Wheelchair Cushion Selection

For selection of wheelchair cushions, the evaluation is quite extensive because cushions are part of the total seating system that also includes the wheelchair. Indeed, no cushion can perform effectively in the prevention of pressure injuries if the wheelchair is not properly fitted. Therefore, it's recommended that a trained seating specialist perform the seat cushion evaluation and selection.

The seating evaluation should also include a mat examination to determine the functional postural limitations of the spine, pelvis, and extremities and to determine appropriate measurements for wheelchair fitting. An extensive functional examination is also required to consider the seating and mobility needs of the individual in the immediate, intermediate, and community environments.[77–79] Individuals with limited mobility who utilize motorized scooters for day-

to-day activities would also benefit from intermittent seating evaluation to identify potential high pressure points. Like wheelchairs, many individuals utilize these devices as a primary means of mobility and independence and may spend a good portion of their day on these surfaces. When taking a patient history, it is a good idea to specifically ask about the amount of time spent sitting on a scooter seat during a 24-hour period, in order to identify this “hidden” risk. The seats of these devices are not categorized like wheelchair cushions and thus may not be evaluated as carefully for pressure redistribution. Current Medicare regulations provide scooter replacement/reimbursement only every 5 years.

Strategies for maintaining tissue integrity can be extremely complicated for spinal cord–injured patients, elderly people, and other populations with degenerative neuromuscular conditions or diseases. For example, a patient’s ability to sit unsupported can be characterized by the amount of external support needed to maintain posture: hands-free, hands-dependent, or prop-sitting with external support only.[80] This capability has significant implications for compensatory functional postures, ability to reposition, and the method used for intermittent pressure relief. Figure 11-11 illustrates three common and effective strategies for intermittent pressure relief.



A

B

C

Figure 11-11. (A) A push-up pressure relief can completely off-load the buttocks but requires sufficient arm strength and trunk control. **(B)** A forward lean can unweight the ischial tuberosities. **(C)** A side-lean pressure relief off-loads the contralateral side and so must be done in both directions.

Specialty Mattress Selection

When selecting a specialty mattress, a patient's weight and the distribution of that weight are important factors. Indeed, each mattress overlay, replacement, or integrated bed unit has weight limits. For heavier patients, bariatric beds should be used. However, for patients approaching the manufacturer's recommended weight limit, the distribution of the weight should be examined. For example, patients who are heavier in the hip region, but who don't exceed the manufacturer's weight limit, may, in fact, need to be placed on a bariatric product in order to achieve effective pressure redistribution. For more information, refer to the good practice statement in the EPUAP/NPIAP/PPPIA guideline on support surfaces for patients with obesity.[77] Patients with contractures may have their weight dispersed unevenly. For example, contractures could pull the heels toward the groin or increase flexion between other body parts creating special needs for tissue management beyond what a mattress can provide. Patients with agitation or restless leg syndrome may be at increased risk of friction injuries, so other skin protectants or offloading devices should be considered.

In addition, patient mobility and activity levels are important consideration. If the goal is to have the patient independently get into and out of bed, this may not be possible with some support surfaces. The patient's needs may also change over time. A therapeutic mattress will not help the person in the chair all day. Therefore, reevaluation of needs as the person's condition changes is important.

Assessing Risk

The most commonly used risk assessment scales for prediction of pressure ulcer incidence are the Braden, Norton, and Waterlow scales.[81–83] The sensitivity and specificity of these scales vary depending on the population setting and the position of the patient's body. For example, different Braden cutoff scores are associated with different settings (nursing home vs. intensive care unit). Risk assessment scales specifically designed for wheelchair users have not been developed.

Bergstrom and colleagues[84] reported that mattress selection based on categorizing patients via a pressure ulcer risk assessment scale produced both efficacious and cost-effective results. Patients of a large tertiary care hospital scoring nine or less on the Braden pressure ulcer risk assessment scale were provided a group 2 support surface (low-air-loss mattress) as a preventive measure. Those scoring above nine were evaluated and provided with the most appropriate surface according to individual patient needs. Results indicated that not only did pressure ulcer incidence and prevalence drop by more than 50% when patients were categorized according to pressure injury risk but costs associated with overlays, replacement mattresses, and low-air-loss beds also decreased.

Similar results have been realized by studies that selected wheelchair cushions from a set of cushion alternatives based on risk assessment. Krouskop and colleagues[85] described how a seating clinic assigned risk to their clients with spinal cord injury by using such factors as gender, interface pressure, lifestyle, and stability. In 80% to 90% of the clients, cushions were selected from three alternatives, with the remaining clients being provided with other cushion types.

When assessing risk, remember that proper follow-up care is necessary to prevent pressure injuries regardless of the cushion or bed prescribed. Skin redness/discoloration often occurs because of positioning and use and doesn't necessarily indicate that a poor surface choice was made.

Clinical Judgment

Clinicians should know how to evaluate bed or cushion performance, which includes an assessment of how adequately the product

provides pressure redistribution, or if the patient is reaching critical immersion or actually having soft tissue in close proximity to the hard undersurface. Clinicians should also observe any powered products for loss of power, sensor malfunctions, or disconnected hoses. Frequently checking the product's performance is especially important for patients who are unable to express their discomfort due to cognitive or communication impairment.

Interface Pressure Mapping

Interface pressure mapping—comparison of a patient's relative responses from one surface to another—can be an effective clinical tool to aid in the selection of a support surface for a specific patient and to monitor the status, including activity of a patient over time. Pressure mapping may also be used to determine the relative effectiveness of modifications to the wheelchair and other positioning devices or to obtain information about pressure relief for patients with spinal cord injuries. For example, Henderson and colleagues used a pressure mapping system to compare three methods of relieving pressure in seated individuals with spinal cord injuries.[86] The positions studied were tilted back 35 degrees, tilted back 65 degrees, and forward-leaning seated posture. The results indicated that the greatest pressure relief over the ischial tuberosities was seen in the forward-leaning position, followed by the 65-degree backward tilt position. Observing the change in pressure distribution on the mapping display allows patients without sensation to observe the effects of various weight-shifting methods and learn to consciously integrate them into their seated behavior. Olney, Vos-Draper and colleagues[87] developed a smart phone pressure map interface for use by wheelchair bound spinal cord injury patients. They found that this innovative technology was well received by users, especially by those using motorized wheelchairs. The smart phone app provides reminders for weight shifting and shows pressure map data in real time.[87]

Using Clinical Practice Guidelines

Clinical practice guidelines offer recommendations, based on scientific evidence and the professional judgment of expert panels, about how healthcare professionals can provide quality care. Several clinical practice guidelines include recommendations on support surface selection: EPUAPNPIAP/PPPIA,[77] WOCN (Wound Ostomy and Continence Nurses Society),[1] and PVA.[88]

How Does the Product Function and How Well Does It Perform?

Answers to what the product does and how it performs should be sought from a variety of sources. Sources of information for support surfaces include marketing materials, controlled clinical trials, and objective indirect data from laboratory testing or clinical studies (interface pressure and other physiological responses).

Laboratory Testing—Mattress and Cushion Performance Standards

Standard laboratory test methods and associated metrics have been developed to describe the performance characteristics of support surfaces. The metrics from these tests are believed to be clinically relevant in pressure injury formation. For example, in 1993, Standards Australia, published methodologies and recommendations for single layer foam mattress characteristics believed to be needed for pressure injury prevention.[89] These characteristics included type of foam (high resiliency), foam density (>2.18 pound/f^3), indentation force deflection (IFD) (40% IFD ≥ 130N), and support factor (65% IFD/25% IFD) (between 1.75 and 2.4). The EPUAP/NPIAP/PPPIA international guidelines[77] recommended that if single layer foam support surfaces are used, they should have these characteristics plus thickness (≥5.9 inches), and moisture vapor transmission (MVTR) (>300 g/m^2/d) should be used for pressure injury prevention. The recommendation was based on the belief that mattresses with these performance characteristics had been shown to be effective over other foam mattresses without these characteristics. The guidelines have a detailed account of the research that led to the recommendation. It is important to realize that, consistent with the research supporting the recommendation, the specifications used to describe the surfaces recommended only apply to solid (single layer) foam mattresses. More work is needed to develop similar recommendations for the numerous other types and variations of support surfaces available on the market today.

The first steps toward developing more generally applicable performance metrics have been taken and are continuing with the publishing of National and International standards for wheelchair seat cushion and mattresses.[10,13] Publishing national or international standards for support surfaces requires that uniform test methods be developed to quantify clinically relevant characteristics. Simply stated, to make valid comparisons among products, characteristics and properties need to be measured using the same test and under the same conditions. Testing conditions should model clinically relevant parameters as closely as possible. The requirement that the tests be repeatable across laboratories in different countries means that standardized tests typically use models rather than human subjects. Clinicians, therefore, must consider test results as relative measures.

The results of standard tests often don't include pass–fail criteria. Just as there are valid reasons to purchase an automobile with an engine that only gets 15 mpg over another with a fuel-efficient engine that gets 25 mpg, so are there reasons to purchase support surfaces with, for example, a lower pressure distribution characteristic but higher moisture dissipation ability over a product with a higher pressure distribution characteristic but lower moisture dissipation ability. The primary objectives of the standard tests are to characterize their different functional properties and to permit comparisons of performance among products with similar functions.

The range of products on the market places a heavy burden on clinicians to keep abreast of new technology; therefore, patients, clinicians, vendors, manufacturers, third-party payers, and researchers all benefit from standard terminology, definitions, and test methods. Clinicians would benefit from a mechanism to objectively match a seat cushion's or support surface's characteristics to the needs of their patients. Vendors benefit by being able to clearly describe products from different manufacturers in a manner understood by clinicians and patients. Testing standards aid manufacturers by guiding new product development and assisting in the redesign of existing products. In addition, standards promote quality assurance within manufacturing processes. The final potential beneficiaries of standards are third-party payers because the seat cushion and support surface market is a payer's market; that is, payer

reimbursement drives the market. A validated system to test and objectively characterize support surfaces will give funding agencies an objective means for making funding decisions. In fact, standards form the basis for certain tests used by funding agencies to classify or categorize products.

Standards for seat cushions are being developed by the ISO and are adopted in the United States by RESNA as an accredited standards organization for the American National Standards Institute. Mattress performance standards development in the United States are organized through NPIAP and RESNA.[10,11] The published standards include tests for a large number of key performance features. For cushions, the tests include metrics for load deflection and hysteresis, envelopment, frictional properties, lateral and forward stiffness, sliding resistance, impact damping, recovery, loaded contour depth and overload deflection, water spillage, heat and water vapor transmissibility, stability of properties with use (fatigue), and biocompatibility.[13] For mattresses, the tests include metrics for immersion, envelopment, heat and water vapor transmissibility, and horizontal stiffness. Although all these tests have clinical relevance, a subset is described below.

Load Deflection

Materials used in mattresses and cushions support the body by compression (foam and air), deflection (gel and viscous fluid), or tension (bladder material and fabric). Material stiffness impacts how materials deform to accommodate the body. Load deflection tests typically involve loading a cushion or support surface with a standardized indenter that mimics a part(s) of the body. Deflection into the support surface is measured as the weight on the indenter is increased.

Clinically, materials can be too stiff or too soft. When too stiff, the body does not immerse, and high pressures or instability can result. When too soft, materials can “bottom out,” leading to poor support and high pressures. Foams are made with different stiffness ratings, called indentation force deflection. Many products use a combination of foam with a softer material positioned on top of a stiffer material.

This configuration permits deflection of the top surface while protecting against critical immersion through the use of the stiffer bottom layer.

Because the amount of air impacts the stiffness of a support surface or cushion, products that use air are often adjustable. Too much air leads to an overly stiff surface, and too little air can lead to critical immersion. In addition to body weight, the amount of tissue, the type of tissue overlying bony prominences (fat, muscle, fascia, edema), and a person's posture or position all influence how stiff a surface must be to adequately support a person.

Immersion and Envelopment

Immersion and envelopment metrics combine to indicate how a support surface is expected to distribute pressure across the contact area of the body. As immersion increases, more and more contact area is available to distribute body weight. However, higher potential for contact area due to deep immersion does not guarantee that pressure across the surface will not concentrate in small areas. The envelopment metric measures how evenly the weight is distributed across the available contact surface. Better envelopment is characterized by a more even distribution of forces.

Frictional Properties

Friction is the result of a relationship between materials; so standardized tests for friction measure the sliding forces of one material on another. In terms of cushions and support surfaces, these standardized tests concentrate on cover materials, such as fabrics and bed linens.

Horizontal Stiffness

The horizontal stiffness metrics measures a support surface's ability to accommodate small shifts of the body across the surface. The force needed to horizontally displace an indenter is measured. A higher peak or final horizontal force may indicate the provision of more stability, and lower forces may indicate enhanced protection

against tissue deformation due to shear forces between the cushion and the body.

Sliding Resistance

Unlike friction tests, which focus on cover material, sliding resistance tests measure the influence of the entire system, including the cover and all support materials. All users of support surfaces and cushions have to transfer onto and off the bed or wheelchair. For people who can't fully transfer, certain materials and designs facilitate easier transfers. However, if a support is too easy to slide on, stability on that surface can be compromised. Standardized tests of sliding resistance involve loading the surface with an indenter modeled on the human body or buttocks and pulling it forward or sideways. The force required to slide the indenter on the surface is measured and reflects sliding resistance.

Loaded Contour Depth and Overload Test

This test measures the immersion of a standardized indenter into a cushion surface. Clinically, the test provides two key pieces of information about seat cushions: the initial contour of a cushion and the amount of deflection that may occur when someone sits on it. The overload portion of the test adds 33% more weight and measures additional immersion of the indenter. A cushion that has "bottomed out" will not deflect further under the additional weight. A support surface should maintain a margin of safety that allows additional cushioning during overload conditions. Certain functional movements and postural adjustments, such as leaning and reaching, impart an overload condition on the surface.

Product Effectiveness

Decades of research have produced moderate and low levels of evidence upon which to base clinical decisions concerning how and when to apply support surfaces for prevention. The Cochrane review of the clinical literature was able to provide only weak statements regarding the relative efficacy of various surface types under specific conditions.[90] The effectiveness of a support service product is

measured by two methods: its efficacy in use by patients and in comparison to similar products. Rather than basing comparisons on functional classification, most studies have compared classes of products based on the product's ability to redistribute pressure.

Considering the limitations ascribed to interface pressure measurement, it may be more useful to categorize devices according to their ability to evenly distribute pressure over the contact surface area rather than ascribe any significance to the magnitude of pressure measured at a particular location. However, the most common comparison has been the use of interface pressure to compare a product against a "standard" hospital bed or mattress. When reviewing this literature, remember that the "standard" support surface probably varies from study to study. Most studies also vary with regard to patient population (e.g., orthopedic or neurologic) and setting studied (e.g., acute care, intensive care, long-term care, or home care). All three independent variables (products compared, subject population, setting) affect both study outcomes and the interpretation of results. Finally, when comparing performance studies, remember that treatment studies, in which subjects already have injuries, are fundamentally different from prevention studies.

Prevention Effectiveness

Generally, studies have shown that nonpowered, reactive support surfaces (foam, air, gel, and combinations of these materials) are more effective in preventing pressure injuries than a "standard" hospital mattress. (The support surface category called "reactive" includes, but is not limited to, surfaces that have been described by the phrase "constant low pressure [CLP]" in the past. The reactive category is broader than the old CLP category because it also contains surfaces with powered features such as low-air-loss). Generalization of the results of studies comparing different pressure redistribution products is difficult. Most comparative studies of various nonpowered reactive products demonstrated no differences in the prevention of pressure injuries compared to each other.[62,90]

Conclusions from research investigating the more complex technology, including low-air-loss and alternating-pressure products,

are similar. Evidence suggests that both low-air-loss and alternating-pressure surfaces are more effective than are “standard” mattresses, but comparative studies of the performance of low-air-loss and alternating-pressure products are inconsistent regarding the clinical superiority of one over the other. Moreover, comparisons of alternating-pressure to constant low-pressure products have not produced definitive differences. Caution should be taken in attempting to generalize the results from these studies because large variations in performance characteristics exist among surfaces within the same category. For example, the abilities of a support surfaces with low-air-loss features to remove moisture from the surface of the product can vary by an order of magnitude.

Because the evidence is not definitive, clinicians must carefully read the original study to generalize the results to a specific clinical situation. When considering a product’s clinical applicability, the characteristics of the population, setting, and products must closely match one’s clinical situation and the limitations of the study must be known. For example, consider the design of alternating-pressure products where such variables as cell height or bladder thickness and cycle timing and frequency can significantly affect product performance. One can’t necessarily generalize the performance of one alternating-pressure product against another. Similarly, the results of a support surface study within acute care might not produce similar results in a home care setting using the same support surfaces.

Evidence about specific cushions and their respective effectiveness is insufficient. Contradictions also exist in the literature regarding the clinical benefits of cushions designed to reduce the risk of sitting-acquired pressure injuries. Most research has used indirect outcomes, such as interface pressure or blood flow. Relatively few studies have measured direct outcomes related to specific types of cushions,[14,33,91,92] but these have not resulted in definitive findings of efficacy of one product over another. One facility performed two studies targeting elderly wheelchair users. The first study found no difference in the incidence of pressure injuries in users of flat foam compared with custom-contoured foam cushions, and a subsequent study found that more users of flat foam (41%) developed injuries compared with users of a contoured foam-viscous fluid cushion

(25%), but this difference did not reach statistical significance. In a study tracking interface pressure and wheelchair cushions in elderly users, Brienza and colleagues[33] found that interface pressures were higher for subjects who developed sitting-acquired pressure injuries compared with those who didn't develop injuries. No definitive relationship was found between interface pressures and cushion types across these subjects.

Brienza[14] performed an RCT in the long-term care setting on 232 nursing home residents comparing pressure injury incidence in participants given a properly sized wheelchair and randomized into a treatment group (n = 113) provided an air cell–based, viscous fluid and foam cushion; or a gel and foam cushion or a control group (n = 119) given a segmented (cross-cut) foam cushion (n = 119). The control group had a significantly greater incidence of ischial tuberosity pressure injuries (6.7% vs. 0.9%, $p = 0.04$).

Treatment Effectiveness

Widely varying subject populations and care settings have complicated the ability to compare results across studies investigating the effectiveness of support surfaces in the treatment of pressure injuries. Furthermore, a number of treatment outcome measures have been used to judge effectiveness, such as the relative or actual reduction in injury size (area or volume), the percentage of injuries healed within a specified time period, and the time until wound closure. Different operational definitions have also been used for wound status, such as "healed" and "closure," making it difficult to compare equitably.

Generally, studies targeting support surfaces for pressure injury treatment have produced results similar to those targeting prevention. Low-air-loss and air-fluidized surfaces have been shown to improve treatment outcomes more effectively compared with "conventional" treatment[93,94] and nonpowered foam alternatives.[95] Results of alternating-pressure surface studies are inconsistent with some studies showing a treatment effect and others showing none. No clinically significant treatment differences have been shown among similar products.

What Other Patient Needs Must Be Met?

While load management and product function are critical areas to consider in matching patient support needs, other pressure redistribution options must also be considered. These options include repositioning, heel protection, managing heel pressure, encouraging ambulation, and managing skin microclimate.

Turning and Repositioning Schedules

The frequency of repositioning required to prevent ischemia is variable and unknown, yet regular repositioning is believed to help deter the deleterious effects of pressure by decreasing the duration of exposure.[96,97] Through the process of repositioning, the body's weight is redistributed, and new pressure areas are introduced. To provide effective pressure relief, both pressure and time must be considered. For example, in 1959, Kosiak recommended that repositioning be done in intervals of 1 to 2 hours based on the interface readings from healthy subjects.[98]

Turning schedules have been studied empirically and experimentally. Bliss[99] studied turning schedules in a spinal injury ward and found that 2 hours was adequate for some, whereas others required more frequent, and some, less frequent, turning. Two important aspects of these findings are that some patients exhibited redness after 2 hours and that many patients disliked frequent turning.

In a paired-facility two-arm (with one-arm crossover) randomized intervention trial, Yap and colleagues[100] studied almost 2,000 residents in 10 Midwest US long-term-care facilities over a 12-month period. Musical cues were played over the facility intercom every 2 hours during the 12-hour daylight period. The music served as a reminder to staff, family members, or residents to reposition residents or move. The primary outcome measure was the frequency of facility-acquired pressure injuries divided by the total number of resident assessments during the study period. Findings showed that intervention facility residents were 45% less likely than comparison facility residents to develop a new PU.

In an experimental study by Knox and colleagues,[97] variables such as temperature, pressure, and redness were monitored while people rested on a mattress for 60, 90, and 120 minutes. Some subjects exhibited redness after each of the intervals, leading the researchers to conclude that a 2-hour turning schedule might not be sufficient.

Such theoretical evidence points directly to the duration of loading as the way to maintain tissue integrity. If, however, the above experiment is only one example of patients experiencing redness after less than a 2-hour turning schedule, then it cannot be construed as the answer for all clinical practices.

While the every-2-hour turn schedule has traditionally been entrenched in clinical practice, new research has challenged this long-held belief. Recent findings from two randomized controlled trials have provided evidence that the frequency of repositioning for persons who are on a viscoelastic foam mattress may be extended for longer than every 2 hours without increasing the incidence of pressure injuries. In a study of 838 nursing home patients who were turned every 4 hours on a viscoelastic mattress, DeFloor[101] reported a reduction in stage 2 and more severe pressure injuries. Vanderwee and colleagues[102] also studied nursing home patients who were also on a viscoelastic foam mattress (n = 235). The time in lateral position of 2 hours was compared with 4 hours in a supine position. Turning these patients every 2 hours did not result in fewer pressure injuries compared with the group turned every 4 hours.

In addition, no knowledge exists as to how turning schedule should be affected by support surfaces. Therefore, the best approach is to evaluate and reevaluate each patient to best determine an appropriate repositioning/turning schedule.

Positioning and Repositioning

According to the EPUAP/NPIAP/EPUAP guidelines[77] for positioning and repositioning individuals in bed a number of recommendations exist in regard to positioning for management of tissue loads.

1. Reposition to relieve or redistribute pressure using manual handling techniques and equipment that do not produce excessive shear stain and shear force.
2. Use the 30-degree side-lying position in preference to the 90 degrees side lying position.
3. Encourage individuals who can reposition themselves to sleep in a 20 to 30 degrees side lying position or flat in bed if not contraindicated.
4. Avoid extended use of prone positioning unless required for management of a medical condition.
5. Keep the head of the bed as flat as possible.

Mimura and colleagues[103] demonstrated that prior to raising the head-of-bed, shear forces on the coccyx can be reduced by first raising the knees. This effect was more pronounced in slender rather than obese body types. See Figure 11-12 for some recommendations. Note the use of pillows and towels to separate and protect bony prominences. Additional positioning techniques include blocking the feet and knees in a flexed position to prevent shear forces created when the patient slides down in bed, foam positioning wedges to offload while maintaining spinal alignment, or fluidized positioners to accommodate kyphosis or scoliosis deformities (Fig. 11-13).

A

B

C

D

Figure 11-12. Horizontal positioning. The following illustrations show how to position patients properly on horizontal surfaces. **(A and B)**30-degree rotation from prone and supine positions, respectively. **(C and D)** Head of the bed elevated 30 degrees or less with unilateral sacral relief and feet blocked, respectively. **(E)** Head of the bed elevated 30 degrees with knees flexed to prevent shearing at the sacrum.

Figure 11-13. Use of moldable fluidized positioner to offload pressure of spinal deformity.

Although there is a common perception that many critically ill patients are “too unstable” to turn or reposition, small shifts in position can be accomplished with careful and incremental movement. These small shifts not only off-load pressure from the skin and bony prominences but may also help reduce the severity of “prolonged gravitational equilibration”.[103,104] For instance, foam wedges or pillows, used to position the patient on his or her side, can be altered slightly every 15 minutes. Pulling them out gradually over 1 to 1½ hours shifts the weight slightly. A patient who is in a wheelchair still requires shifts in his or her body weight. If possible, this patient should be taught to shift his or her weight every 15 minutes with repositioning every hour (see Fig. 11-11).

Heel Protection

The 2019 EPUAP/NPIAP/PPPIA guidelines[77] address in detail differentiation in treatment recommendations to vulnerable areas of skin and soft tissue based upon the distinct characteristics of each area. The heel is such an area that, by virtue of its structure, necessitates particular attention. The small radius of curvature of the calcaneus corresponds to a small area of contact with any support surface. The thin layer of subcutaneous tissue under the skin can be subjected to increased pressure from the weight of the foot with the addition of the weight of portions of the lower limb depending on how the leg is positioned. Considering that the lower limb holds about one-sixth of total body weight, if even a small percentage of the weight of the lower limb is resting on the heel, the resultant increased pressure will be directed to a small area of tissue with little innate protection. Even air support systems will not be able to prevent the resultant high interface pressure. That is why the NPIAP/EPUAP/PPPIA guidelines[77] recommend the use of additional pressure-reducing devices for immobile patients' heels.

The goal of heel suspension devices recommended by the EPUAP/NPIAP/PPPIA clinical practice guidelines[77] is to off-load the heel. For prone patients, this should be done in such a way as to elevate the heel above the bed surface and preclude pressure being placed on the Achilles tendon or the popliteal vein. There are a variety of common and specialized means for accomplishing this task. Pillows are among the most commonly available resources, but they are less than ideal. While standard pillows perform the prescribed function of off-loading the heel, they do not prevent footdrop and must be repeatedly and assiduously repositioned to maintain optimal suspension (Fig. 11-14) as they have the tendency to shift due to patient movement or weight. A better option would be fluidized positioners. These devices can be employed for offloading since they are not easily displaced, can be molded against a limb, and have been shown to effectively redistribute pressure.

Figure 11-14. Heel suspension boot with adjustable foot drop strap.

Compounding the issue of pressure-induced soft tissue damage to the heel is the degree of discomfort such injury presents to the patient. Heel injuries can be particularly painful and slower, if not outright difficult, to heal. The resulting damage can escalate to the point of infection and even, in gravest conditions, amputation of the

foot. The consequences of heel pressure injuries go beyond the discomfort of the patient. The costs attached to said injury rise proportionally with morbidity and length of hospital stay, emphasizing the need to address prevention of heel injury in high-risk patients.

Evidence for Managing Heel Pressure

While not a primary focus of studies on pressure injury prevention, protecting the heel from injury has been addressed on a small scale in research studies. Published studies have examined the pressure distribution capabilities of heel protection devices including wraps, dressings, pillows, water-filled gloves, and other specialty products using interface pressure or pressure injury incidence as primary outcomes.[105,106]

The functionality of products that offer protection to the heel is based upon the goal of optimizing therapeutic function, much as with any support surface relative to the body part, it is designed to bear. Most products designed to protect the heel are constructed using a combination of materials to offer both padding and support and incorporating more than one strategy to distribute pressure across the heel support surface. The efficacy of the device depends on intrinsic and extrinsic factors, including but not limited to the mechanical properties of the heel tissue and the device, how well the device conforms to the shape of the heel, and how weight is distributed in the heel. More ideal pressure distribution will occur when there is relatively little change to the shape of the heel in its loaded condition as compared to its unloaded state.[107]

Direct pressure on the heel is a major contributor to pressure injury, and so off-loading of the heels is a relatively straightforward and effective intervention for at-risk patients. However, another factor to consider is the role that shear stress can play in exacerbating damage to vulnerable tissue. Shear stress on the heel can occur from a number or sources including repeated active or passive shifting in bed, agitated movement, or even acceleration/deceleration forces in a moving vehicle. Safe patient handling techniques and the use of prophylactic multilayer foam heel dressings have been shown to be effective in reducing shear stress.[105,108] Zernike designed a study of

the efficacy of clinically known heel devices and procedures. He compared the preventive use of routine nursing care, hydrocolloid dressings, egg-crate foam, polyester-filled heel boots, and foam footdrop splints in geriatric, orthopedic patients at risk for pressure injuries.[107] A second study combined 4″ × 4″ gauze pads with an absorbent pad held in place by a gauze roll and compared the results with those obtained by use of a laminated foam boot.[102,109] Although the statistical analyses were limited in both studies, the most effective devices for preventing pressure injuries of the heel were determined to be the egg-crate boot and the laminated foam boot. The other preventative methods studied were less effective either because they resulted in an increased incidence of pressure injury or, in the case of footdrop splints, were too uncomfortable for sustained use despite effectively suspending the heel. A notable observation in Zernike's study[107] is that heel tissue deteriorated despite routine nursing care (heel observation every 2 to 3 hours and direct pressure relief and repositioning of patients' heels). A similar study was able to achieve zero incidence of pressure injuries in 30 hip replacement patients. The latter study implemented prompt reporting of heel discomfort by patients to the nursing staff, with subsequent simultaneous use of heel elevation with a pillow or bath blanket under the calf and a quilted heel protector.[109]

Flemister[110] also examined heel interface pressures in a comparison of the use of a foam versus polyester heel protector. The study of seven subjects, patients assessed to be at moderate-to-high risk for pressure ulcer development, demonstrated the greater efficacy of foam in reducing pressure on the heel. The foam heel protector marginally reduced heel interface pressures (1.3 mm Hg), whereas, in contrast, the polyester protector resulted in a significant increase in interface pressure. Some studies have demonstrated that multilayer silicone dressings used on the heels are effective in preventing pressure injuries by reducing friction and shear stress.[105] While there are a variety of heel protective devices available, their use must be tailored to the individual patient characteristics and circumstances. The EPUAP/NPIAP/PPPIA clinical practice

guidelines[77] recommend that prophylactic dressings be used as adjuncts to heel offloading and other strategies.

What's Available in This Setting?

A patient's needs can change over the course of his or her illness. A product that's appropriate for acute care use may not work in the patient's home. Clinical judgment should consider therapy goals (ease of getting into or out of the bed for therapy or mobility), ability of the patient to move himself or herself in the bed, other complications (heel breakdown, pulmonary complications), and body weight and weight distribution.

What's Practical?

The issue of practicality relates to the overall care plan, the goals of load management (prevention vs. treatment), how complicated the product is to use, and whether it will be operated by a healthcare professional, family member, or the patient. Many hospitals, long-term care facilities, and home care agencies have developed product selection guidelines that are usually presented as an algorithm or graph. These guidelines are typically based on the availability of equipment from previous purchases in accordance with other published guidelines. Another common method used in selection and purchasing decisions is the subjective assessment of equipment based on a trial use in the clinical setting. This gives the staff a chance to use the equipment in a variety of situations and judge its performance.

How Easy Is the Product to Use?

Equipment that's easy to use has been associated with successful compliance. Awkward design or difficult assembly may cause the patient and his or her family to abandon using the equipment or increase the likelihood of misuse. Directions must be clear and obvious. For example, if the product is powered, it should have a battery backup to facilitate transferring patients to other locations as needed.

What Service and Maintenance Is Available?

A 24-hour call service with on-site repair or replacement is essential.

What Type of Alarm System Does the Product Have?

Visual alarms are rarely sufficient, especially if the alarm is obscured under the bed. In the home-care setting, visible alarms are of little value unless there's constant attendance.

Is the Equipment Easily Maintained?

If the equipment must be deflated for storage, reinflation time may be critical as well as the ability to deflate the equipment in the event of a cardiac arrest. Additionally, if the product isn't a personal use item, one must consider how long it takes to clean and ready the item for subsequent use.

What Operating Mechanism and Space Requirements Does the Product Have?

If the product has some form of movement, is cessation possible to facilitate such procedures as bedpan usage and hygiene care? Is the product the correct size for the home or setting where it will be used? Is the floor structure sound and capable of holding the weight of the bed? Will the bed fit through the door?

How Much Does the Product Cost?

Technology has produced some innovative treatment products, but costs are high for patients, insurance companies, and hospitals. Prevention is more cost-effective than is any other treatment. Available technology (support surfaces) may provide solutions when used correctly by properly educated staff. Indeed, one recent study demonstrated that educated staff can reduce the number of pressure injuries better than noneducated staff. The same study indicated that the cost of care is significantly less under the supervision of educated staff.[111]

The cost of support surfaces varies. Third-party reimbursement (Medicare may provide coverage), rental versus purchase, and cost–benefit issues need to be evaluated carefully. Some devices are only available on a rental basis. Although reimbursement is an issue in any setting, operational costs are of particular importance if the product is used in the home care setting where costs may be absorbed by the patient.

Consideration should also be given to the projected number of days that a support surface will be in use. More patients are being placed on advanced technology surfaces for considerably longer periods of time. Cost-effectiveness may be measured by relating the cost of the product to its efficacy.

Practice Point

Consider these patient factors before purchasing support surfaces:

- Ease of use
- Operational costs of the equipment
- Service contracts and backup service
- Alarm systems
- Daily maintenance
- Operating mechanisms and space requirements

When managing the allocation of specialty mattress and bed systems, a continuous process of evaluation and reevaluation should be employed to ensure that the patient's needs are reassessed on a regular basis.

Summary

Understanding the nature of pressure injury etiology, the factors affecting pressure redistribution, and other physical factors associated with the use of specific support surface products and heel protection products is a necessity for nursing and other healthcare personnel involved with tissue integrity management. As standardized test methods are developed and used for support surface products, the clinical validation of specialized protective devices and the development of more specific clinical practice guidelines will be possible. Until then, matching products to patient needs is a challenging process and must be based on the available evidence regarding the performance characteristics of existing support surfaces. Continued research in heel protection is needed.

Heels in patients at risk for pressure injuries require additional protection beyond the use of specialty beds and mattress overlays. It is important to keep in mind that pillows don't protect against footdrop, require time and attention to positioning details, and can't protect the heels continuously due to patient movement. To provide continuous heel suspension via heel protective devices, clinicians must consider proper fit, turning schedules and number of turning surfaces, patient position, the presence of additional equipment, and the performance characteristics of the product when selecting heel protection devices (see Fig. 11-14).

PATIENT SCENARIO

Clinical Data

Mr. P. is a 76-year-old male admitted to the ICU following trans-catheter replacement of the aortic valve. Mr. P. is a retired chemist and is accompanied by his wife (a retired RN) who has been providing care at home. An unstageable PI on the sacrum is noted on admission, as well as a neuropathic ulcer on the 5th metatarsal of

the right foot. Mr. P's wounds have been managed at a local Wound Center over the past 6 weeks with improvement.

Past medical history is significant for peripheral vascular disease (primarily arterial), diabetes mellitus, acute renal failure, mitral valve regurgitation, and kyphosis with protruding spinous processes. Mr. P. also has chronic shoulder and hip pain as a result of a motor vehicle crash many years ago. His acute and chronic pain makes finding a comfortable and functional position difficult, and he states that he cannot lay flat for any appreciable amount of time. He is able to walk at home for a distance of approximately 60 feet using a single point cane. He wears an unmodified Darco shoe to off-load pressure from his foot. Due to his long-standing diabetes and peripheral vascular disease, Mr. P. states that he cannot feel his feet.

You have been asked to make recommendations for an appropriate support surface to promote healing of his sacral pressure injury, prevent further skin breakdown, as well as develop strategies to promote postoperative progressive mobility.

Because of Mr. P's overall debility and increased risk for PI development (Braden total score: 17), he was placed on a powered reactive pressure redistribution mattress. A fluidized positioner was used to offload pressure from his spine and sacrum because it could be comfortably molded around his kyphotic spinal column. Multilayer silicone dressings were placed over the protruding spinous processes and sacrum to protect from shear and friction. While in bed, heels were gently offloaded with vascular boots (due to significant arterial disease) to protect from inadvertent trauma and provide warmth. Physical therapy was consulted for other positioning and mobilization suggestions. PT adjusted the Darco foot orthotic to off-load pressure from the neuropathic ulcer, and both the patient and his wife were instructed that the orthotic shoe needed to be worn whenever out of bed and walking. When out of bed to the chair, an offloading cushion with a "V" coccyx cut-out was used to provide better pelvic stability and no contact or shear stress on the sacrum.

Case Discussion

The complexity of Mr. P's illness and multiple comorbidities required a multidisciplinary approach to his postoperative recovery. Listening to and involving both the patient and his wife were very helpful in creating a plan that would work both in the hospital and when transitioning home. Although his pressure injury and neuropathic ulcer did not seem to be a high priority issue in the early postoperative period, they both contributed to his perceived quality of life and ability to mobilize and walk. The short-term objectives toward the overall goal of returning home were: Improve overall comfort, improve positioning and support both in and out of bed, and modify the foot orthotic to help healing of the neuropathic ulcer, and prevent further tissue damage in an insensate foot.

Show What You Know

1. **The feature of a support surface primarily indicated for pulmonary therapy that provides rotation about a longitudinal axis as characterized by degree of patient turn, duration, and frequency is called:**
   A. low-air-loss.
   B. alternating pressure.
   C. air-fluidized.
   D. lateral rotation.

2. **The selection of an appropriate support surface depends on:**
   A. the clinical condition of the patient.
   B. the characteristics of the support surface.
   C. the characteristics of the care setting.
   D. all of the above.

3. **Support surfaces are designed to:**
   A. lower pressure.
   B. eliminate pressure.
   C. redistribute pressure.
   D. relieve pressure.

4. **The ability of a support surface to distribute load over the contact areas of the human body is:**
   A. immersion.
   B. envelopment.
   C. pressure gradient.
   D. pressure redistribution.

Answers to these questions are found on pages 683 and 684.

References

1. McNichol, L., Watts, C., Mackey, D., et al. Identifying the Right Surface for the Right Patient at the Right Time: Generation and Content Validation of an Algorithm for Support Surface Selection. Journal of Wound Ostomy & Continence Nursing 42(1):19-37, 2015.
2. Bergstrom, N., Braden, B., Kemp, M., et al. Predicting Pressure Ulcer Risk: A Multisite Study of the Predictive Validity of the Braden Scale. Nursing Research 47(5):261-9, 1998.
3. Gadd, M. M. Preventing Hospital-acquired Pressure Ulcers: Improving Quality of Outcomes by Placing Emphasis on the Braden Subscale Scores. Journal of Wound Ostomy & Continence Nursing 39(3):292-4, 2012.
4. Gadd, M. M. Braden Scale Cumulative Score Versus Subscale Scores: Are We Missing Opportunities for Pressure Ulcer Prevention? Journal of Wound Ostomy & Continence Nursing 41(1):86-9, 2014.
5. Menegon, D. B., Bercini, R. R., Santos, C. T. D., et al. Braden Subscales Analysis as Indicative of Risk for Pressure Ulcer. Texto & Contexto-Enfermagem 21(4): 854-61, 2012.
6. Tescher, A. N., Branda, M. E., Byrne, T. O., et al. All At-risk Patients Are Not Created Equal: Analysis of Braden Pressure Ulcer Risk Scores to Identify Specific Risks. Journal of Wound Ostomy & Continence Nursing 39(3):282-91, 2012.
7. Bergquist, S. Subscales, Subscores, or Summative Score: Evaluating the Contribution of Braden Scale Items for Predicting Pressure Ulcer Risk in Older Adults Receiving Home Health Care. Journal of Wound Ostomy & Continence Nursing 28(6):279-89, 2001.
8. Cox, J. Predictive Power of the Braden Scale for Pressure Sore Risk in Adult Critical Care Patients: A Comprehensive Review. Journal of Wound Ostomy & Continence Nursing 39(6):613-21, 2012.
9. Krouskop, T., Van Rijswijk, L. Standardizing Performance-based Criteria for Support Surfaces. Ostomy/Wound Management 41(1):34-6, 38, 40-35, 1995.
10. RESNA. *American National Standard for Support Surfaces—Volume 1: Requirements and Test Methods for Full Body Support Surfaces (ANSI/RESNA SS-1:2019)*. 2019. Available at: https://www.resna.org/standards/support-surfaces/support-surfaces
11. National Pressure Ulcer Advisory Panel. Support Surface Standards Initiative —Terms and Definitions. 2019.
12. (S3I), S. S. S. I. Available at: https://www.npuap.org/resources/educational-and-clinical-resources/support- surface-standards-initiative-s3i/, 2019.
13. ISO, I. O. o. S. *Wheelchair Seating—Part 2: Determination of Physical and Mechanical Characteristics of Devices Intended to Manage Tissue Integrity—Seat Cushions*. 2018.

14. Brienza, D., Kelsey, S., Karg, P., et al. A Randomized Clinical Trial on Preventing Pressure Ulcers with Wheelchair Seat Cushions. Journal of the American Geriatrics Society 58(12):2308-14, 2010.
15. Cochran, G. “Identification and Control of Biophysical Factors Responsible for Soft Tissue Break Down.” *RSA Progress Report*, 1979.
16. Silver-Thorn, M.B. “In Vivo Indentation of Lower Extremity Limb Soft Tissue.” IEEF Transactions on Rehabilitation Engineering 7(3):268-77, 1999.
17. Kosiak, M., Kubicek, W. G., Olson, M. Evaluation of pressure as factor in the production of ischial ulcers. Archives of Physical Medicine and Rehabilitation 39:623-9, 1958.
18. Reswick, J., Rodgers, J. Experience at Rancho Los Amigos Hospital with Devices and Techniques to Prevent Pressure Sores. In: Bedsore Biomechanics. Baltimore, MD: University Park Press, 1975.
19. Reswick, J., Rogers, J. Experience at Rancho Los Amigos Hospital with Devices and Techniques to Prevent Pressure Sores. In: Bedsore Biomechanics. New York, NY: Springer, 1976:301-10.
20. Reswick, J. B., Rogers, J. E. Experience at Rancho Los Amigos hospital with Devices and Techniques to Prevent Pressure Sores. In R. M. Kenedi, J. M. Cowden, J. T. Scales, eds. Bedsore Biomechanics. Baltimore, MD: University Park Press, 1976:301-10.
21. Sprigle, S., Sonenblum, S. Assessing Evidence Supporting Redistribution of Pressure for Pressure Ulcer Prevention: A Review. Journal of Rehabilitation Research and Development 48(3):203-13, 2011.
22. Bouten, C. V., Knight, M. M., Lee, D. A., et al. Compressive Deformation and Damage of Muscle Cell Subpopulations in a Model System. Annals of Biomedical Engineering 29(2):153-63, 2001. Available at: C:\Jan_PDF\Bouten_2001_MuscleCellDamage.pdf
23. Tsuji, S., Ichioka, S., Sekiya, N., et al. Analysis of Ischemia-reperfusion Injury in a Microcirculatory Model of Pressure Ulcers. Wound Repair and Regeneration 13(2):209-15, 2005.
24. Breuls, R. G. M., Bouten, C. V. C., Oomens, C. W. J., et al. Compression Induced Cell Damage in Engineered Muscle Tissue: An in Vitro Model to Study Pressure Ulcer Aetiology. Annals of Biomedical Engineering 31:1357-64, 2003. Available at: C:\Jan_PDF\Breuls_2003_CellDamagePU.pdf
25. Linder-Ganz, E., Engelberg, S., Scheinowitz, M., et al. Pressure–time Cell Death Threshold for Albino Rat Skeletal Muscles as Related to Pressure Sore Biomechanics. Journal of Biomechanics 39(14):2725-32, 2006.
26. Oomens, C., Loerakker, S., Bader, D. The Importance of Internal Strain as Opposed to Interface Pressure in the Prevention of Pressure Related Deep Tissue Injury. Journal of Tissue Viability 19(2):35-42, 2010.
27. McCance, K., Huenter, S. Pathophysiology. The Biologic Basis for Disease in Adults and Children, 7th ed. St. Louis, MO: Mosby, 2014.

28. Oomens, C. W., Bader, D. L., Loerakker, S., et al. Pressure Induced Deep Tissue Injury Explained. Annals of Biomedical Engineering 43(2):297-305, 2015.
29. Gefen, A., van Nierop, B., Bader, D. L., et al. Strain-time Cell-death Threshold for Skeletal Muscle in a Tissue-engineered Model System for Deep Tissue Injury. Journal of Biomechanics 41(9):2003-12, 2008.
30. Farid, K. J., Winkelman, C., Rizkala, A., et al. Using Temperature of Pressure-related Intact Discolored Areas of Skin to Detect Deep Tissue Injury: An Observational, Retrospective, Correlational Study. Ostomy/Wound Management 58(8):20-31 2012.
31. Sae-Sia, W., Wipke-Tevis, D. D., Williams, D. A. Elevated Sacral Skin Temperature (T(s)): A Risk Factor for Pressure Ulcer Development in Hospitalized Neurologically Impaired Thai Patients. Applied Nursing Research 18(1):29-35, 2005.
32. Yusuf, S., Okuwa, M., Shigeta, Y., et al. Microclimate and Development of Pressure Ulcers and Superficial Skin Changes. International Wound Journal 12(1):40-6, 2015.
33. Brienza, D. M., Karg, P. E., Geyer, M. J., et al. The Relationship between Pressure Ulcer Incidence and Buttock-seat Cushion Interface Pressure in at-risk Elderly Wheelchair Users. Archives of Physical Medicine and Rehabilitation 82(4):529-33, 2001. Available at: C:\Jan_PDF\Brienza_2001_PUIncidencePressure.pdf
34. Maurer, C. L., Sprigle, S. Effect of Seat Inclination on Seated Pressures of Individuals with Spinal Cord Injury. Physical Therapy 84(3):255-61, 2004.
35. Sakai, K., Sanada, H., Matsui, N., et al. Continuous Monitoring of Interface Pressure Distribution in Intensive Care Patients for Pressure Ulcer Prevention. Journal of Advanced Nursing 65(4):809-17, 2009.
36. Sonenblum, S. E., Vonk, T. E., Janssen, T. W., et al. Effects of Wheelchair Cushions and Pressure Relief Maneuvers on Ischial Interface Pressure and Blood Flow in People with Spinal Cord Injury. Archives of Physical Medicine and Rehabilitation 95(7):1350-7, 2014.
37. Conine, T. A., Hershler, C., Daechsel, D., et al. Pressure Ulcer Prophylaxis in Elderly Patients using Polyurethane Foam or Jay Wheelchair Cushions. International Journal of Rehabilitation Research 17(2):123-37, 1994.
38. Drummond, D., Breed, A. L., Narechania, R. Relationship of Spine Deformity and Pelvic Obliquity on Sitting Pressure Distributions and Decubitus Ulceration. Journal of Pediatric Orthopedics 5(4): 396-402, 1985.
39. Pipkin, L., Sprigle, S. Effect of Model Design, Cushion Construction, and Interface Pressure Mats on Interface Pressure and Immersion. Journal of Rehabilitation Research and Development 45(6):875-82, 2008.
40. Krouskop, T. A. A Synthesis of the Factors that Contribute to Pressure Sore Formation. Medical Hypotheses 11(2):255-67, 1983.

41. Reddy, N. P., Cochran, G. V. B., Krouskop, T. A. Interstitial Fluid Flow as a Factor in Decubitus Ulcer Formation. Journal of Biomechanics 14(12):879-81, 1981.
42. Nemoto, T., Kubota, R., Murasawa, Y., et al. Viscoelastic Properties of the Human Dermis and Other Connective Tissues and Its Relevance to Tissue Aging and Aging–Related Disease. In: J. De Vicente, ed. Viscoelasticity-from Theory to Biological Applications 2012:157-70.
43. Sopher, R., Gefen, A. Effects of Skin Wrinkles, Age and Wetness on Mechanical Loads in the Stratum Corneum as Related to Skin Lesions. Medical & Biological Engineering & Computing 49(1):97-105, 2011.
44. Derler, S., Gerhardt, L.-C. Tribology of Skin: Review and Analysis of Experimental Results for the Friction Coefficient of Human Skin. Tribology Letters 45(1): 1-27, 2012.
45. Rotaru, G.-M., Pille, D., Lehmeier, F., et al. Friction Between Human Skin and Medical Textiles for Decubitus Prevention. Tribology International 65:91-6, 2013.
46. Lachenbruch, C., Tzen, Y.-T., Brienza, D. M., et al. The Relative Contributions of Interface Pressure, Shear Stress, and Temperature on Tissue Ischemia: A Cross-sectional Pilot Study. Ostomy/Wound Management 59(3):25-34, 2013.
47. Manorama, A., Meyer, R., Wiseman, R., et al. Quantifying the Effects of External Shear Loads on Arterial and Venous Blood Flow: Implications for Pressure Ulcer Development. Clinical Biomechanics 28(5):574-8, 2013.
48. Ohura, T. A New Treatment and Care of Pressure Ulcers-from the View of External Forces (Inadequate Nursing Care): Part 1: External Force and its Clinical Influence-the Relationships Between Fundamental Biomechanics and Clinical Findings. World Council of Enterostomal Therapists Journal 33(2):14, 2013.
49. Brown, A., Brengelmann, G. Energy Metabolism. Philadelphia, PA: W.B. Saunders Co., 1965
50. Johnson, J. M., O'Leary, D. S., Taylor, W. F., et al. Reflex Regulation of Sweat Rate by Skin Temperature in Exercising Humans. Journal of Applied Physiology 56(5):1283-8, 1984.
51. Johnson, J. M., Park, M. K. Reflex Control of Skin Blood Flow by Skin Temperature: Role of Core Temperature. Journal of Applied Physiology 47(6):1188-93, 1979.
52. Aronovitch, S. A., Wilber, M., Slezak, S., et al. A Comparative Study of an Alternating Air Mattress for the Prevention of Pressure Ulcers in Surgical Patients. Ostomy/Wound Management 45(3):34-40, 1999.
53. Patel, S., Knapp, C. F., Donofrio, J. C., et al. Temperature Effects on Surface Pressure-induced Changes in Rat Skin Perfusion: Implications in Pressure Ulcer Development. Journal of Rehabilitation Research and Development 36(3):189-201, 1999.

54. Gefen, A. How Do Microclimate Factors Affect the Risk for Superficial Pressure Ulcers: A Mathematical Modeling Study. Journal of Tissue Viability 20(3): 81-8, 2011.
55. Liao, F., O'Brien W.D. Jr, Jan, Y.-K. Assessing Complexity of Skin Blood Flow Oscillations in Response to Locally Applied Heating and Pressure in Rats: Implications for Pressure Ulcer Risk. Physica A: Statistical Mechanics and its Applications 392(20):4905-15, 2013.
56. Kokate, J. Y., Leland, K. J., Held, A. M., et al. Temperature-modulated Pressure Ulcers: A Porcine Model. Archives of Physical Medicine and Rehabilitation 76(7):666-73, 1995.
57. Iaizzo, P. A. Temperature Modulation of Pressure Ulcer Formation: Using a Swine Model. Wounds 16(11):336-43, 2004.
58. Tzen, Y.-T., Brienza, D. M., Karg, P., et al. Effects of Local Cooling on Sacral Skin Perfusion Response to Pressure: Implications for Pressure Ulcer Prevention. Journal of Tissue Viability 19(3):86-97, 2010.
59. Tzen, Y.-T., Brienza, D. M., Karg, P. E., et al. Effectiveness of Local Cooling for Enhancing Tissue Ischemia Tolerance in People with Spinal Cord Injury. The Journal of Spinal Cord Medicine 36(4):357-64, 2013. doi:10.1179/2045772312Y.0000000085
60. Shaked, E., Gefen, A. Modeling the Effects of Moisture-related Skin-Support Friction on the Risk for Superficial Pressure Ulcers During Patient Repositioning in Bed. Frontiers in Bioengineering and Biotechnology 1:9, 2013.
61. Wildnauer, R. H., Bothwell, J. W., Douglass, A. B. Stratum Corneum Biomechanical Properties I. Influence of Relative Humidity on Normal and Extracted Human Stratum Corneum. Journal of Investigative Dermatology 56(1):72-8, 1971.
62. McInnes, E., Jammali-Blasi, A., Bell-Syer, S. E. M., et al. Support Surfaces for Pressure Ulcer Prevention. The Cochrane Database of Systematic Reviews 4:CD001735, 2011.
63. McInnes, E., Jammali-Blasi, A., Bell-Syer, S. E. M., et al. Support Surfaces for Pressure Ulcer Prevention [DE-SR]. Cochrane Database of Systematic Reviews 9:CD001735, 2015.
64. Call, E., Jones, R., DeMonja, K., et al. "The Elastic Limit": Introducing a Novel Concept in Communicating Excessive Shear and Tissue Deformation. World Council of Enterostomal Therapists Journal 37(4):16-20, 2017.
65. Wheelchair and Related Seating Standards Committee. Available at: http://www.resna.org/standards/wheelchair- seating/wheelchair-and-related-seating
66. Noble, P., Goode, B., Krouskop, T., et al. The Influence of Environmental Aging Upon the Loadbearing Properties of Polyurethane Foams. Journal of Rehabilitation Research and Development 21(2):31-8, 1984.

67. Stewart, S., Palmieri, V., Cochran, G. Wheelchair Cushion Effect on Skin Temperature, Heat Flux, and Relative Humidity. Archives of Physical Medicine and Rehabilitation 61(5):229-33, 1980.
68. Wells, J., Karr, D. Interface Pressure, Wound Healing, and Satisfaction in the Evaluation of a Non-powered Fluid Mattress. Ostomy/Wound Management 44(2): 38-42, 44-36, 48 passim, 1998.
69. Thompson, P., Anderson, J., Langemo, D., et al. Support Surfaces: Reducing Pressure Ulcer Risk. Nursing Management 40(10):49-51, 2009.
70. Holzapfel, S. Support Surfaces and Their Use in the Prevention and Treatment of Pressure Ulcers. Journal of ET Nursing 20(6):251-60, 1992.
71. American National Standards Institute (ANSI), & Rehabilitation Engineering and Assistive Technology Society of North America (RESNA). *RESNA-SS1: Volume 1: Requirements and Test Methods for Full Body Support Surfaces SS-1: Section 1 Vocabulary; Section 2 Standard Protocol for Measuring Immersion in Full Body Support Surfaces; Section 3 Standard Protocol for Measuring Heat and Water Vapor Dissipation Characteristics of Full Body Support Surfaces - Body Analog Method; Section 4 Standard Protocol for Measuring Heat and Water Vapor Dissipation Characteristics of Full Body Support Surfaces - Sweating Guarded Hot Plate (SGHP) Method; Section 5 Support Surface Horizontal Stiffness Test ; Section 6 Envelopment and Immersion – Hemispherical Indenter Test; Section 7 Envelopment with Dual Semispherical Indenter Test; Section 8 Standard Protocol for Measuring Water Vapor Dissipation Characteristics of Full Body Support Surfaces – Heated Water Bladder Method.* Arlington, VA: RESNA, 2018.
72. VanGilder, C., Lachenbruch, C. A. Air-fluidized Therapy: Physical Properties and Clinical Uses. Annals of Plastic Surgery 65(3):364-70, 2010.
73. Gunther, R., Brofeldt, B. Increased Lymphatic Flow: Effect of a Pulsating Air Suspension Bed System. Wounds 8(4):134-40, 1996.
74. Karg, P., Ranganathan, V. K., Churilla, M., et al. Sacral Skin Blood Flow Response to Alternating Pressure Operating Room Overlay. Journal of Tissue Viability 28(2):75-80, 2019.
75. Won, B.-H., Song, C.-S. Alternating Pressure Profile Characteristics of Powered Pressure Ulcer Preventing Devices. Journal of the Ergonomics Society of Korea 29(4):639-46, 2010.
76. Anderson, C., Rappl, L. Lateral Rotation Mattresses for Wound Healing. Journal of Wound Ostomy & Continence Nursing 30(3):S23-4, 2003.
77. European Pressure Ulcer Advisory Panel, National Pressure Injury Advisory Panel, and Pan Pacific Pressure Injury Alliance. In: E. Haesler, ed. Prevention and Treatment of Pressure Ulcers/Injuries. EPUAP/NPIAP/PPPIA, 2019.
78. Engström, B. Seating for Independence. Ergonomic Seating and Propulsion Improves Performance. Stockholm, Sweden: Posturalis, 1997.

79. Waugh, K. Therapeutic Seating I: Principles and Assessment. Arlington, VA: Rehabilitation Engineering and Assistive Technology Society of North America, 1997
80. Minkel, J. L. Seating and Mobility Considerations for People with Spinal Cord Injury. Physical Therapy 80(7):701-9, 2000.
81. Braden, B. J., Bergstrom, N. Clinical Utility of the Braden Scale for Predicting Pressure Sore Risk. Advances in Skin & Wound Care 2(3):44-51, 1989.
82. Braden, B. J., Bergstrom, N. Predictive Validity of the Braden Scale for Pressure Sore Risk in a Nursing Home Population. Research in Nursing & Health 17(6):459-70, 1994.
83. Norton, D. Norton Scale for Decubitus Prevention. Krankenpflege (Frankfurt am Main, Germany) 34(1):16, 1980.
84. Bergstrom, N., Braden, B., Boynton, P., et al. Using a Research-based Assessment Scale in Clinical Practice. The Nursing Clinics of North America 30(3):539-51, 1995.
85. Krouskop, T. A., Cullen, B., Garber, S. L. Custom Selection of Support Surfaces for Wheelchairs and Beds: One Size Doesn't Fit All. Dermatology Nursing 4(3):191-4, 1992.
86. Henderson, J. L., Price, S. H., Brandstater, M. E., et al. Efficacy of Three Measures to Relieve Pressure in Seated Persons with Spinal Cord Injury. Archives of Physical Medicine and Rehabilitation 75(5):535-9, 1994.
87. Olney, C. M., Vos-Draper, T., Egginton, J., et al. Development of a Comprehensive Mobile Assessment of Pressure (CMAP) System for Pressure Injury Prevention for Veterans with Spinal Cord Injury. The Journal of Spinal Cord Medicine 42(6):685-94, 2019.
88. Consortium for Spinal Cord Medicine Clinical Practice Guidelines Pressure Ulcer Prevention and Treatment Following Spinal Cord Injury: A Clinical Practice Guideline for Health-care Professionals. The Journal of Spinal Cord Medicine 24:S40, 2001.
89. Committee PL/36 on Flexible Polyurethane, Australian Standard® AS2281-1993. Flexible Cellular Polyurethane for Seat Cushioning and Bedding. Homebush, NSW: Standards Australia (Standards Association of Australia), 1993.
90. Moore, Z., Haynes, J. S., Callaghan, R. Prevention and Management of Pressure Ulcers: Support Surfaces. British Journal of Nursing 23(Suppl 6):S36-43, 2014.
91. Geyer, M. J., Brienza, D. M., Karg, P., et al. A Randomized Control Trial to Evaluate Pressure-reducing Seat Cushions for Elderly Wheelchair Users. Advances in Skin & Wound Care 14(3):120-32, 2001.
92. Trewartha, M., Stiller, K. Comparison of the Pressure Redistribution Qualities of Two Air-filled Wheelchair Cushions for People with Spinal Cord Injuries. Australian Occupational Therapy Journal 58(4):287-92, 2011.

93. Black, J., Berke, C., Urzendowski, G. Pressure Ulcer Incidence and Progression in Critically Ill Subjects: Influence of Low Air Loss Mattress Versus A Powered Air Pressure Redistribution Mattress. [T Support Surfaces, Critical Care]. Journal of Wound, Ostomy, and Continence Nursing 39(3):267-73, 2012. doi: 10.1097/WON.0b013e3182514c50.
94. Johnson, J., Peterson, D., Campbell, B., et al. Hospital-acquired Pressure Ulcer Prevalence—Evaluating Low-Air-Loss Beds [T Support Surfaces-Prevention, Overlap with P & I]. Journal of Wound, Ostomy, and Continence Nursing 38(1):55-60, 2011. Available at: https://library1.unmc.edu/login?url=http://search.ebscohost.com/login.aspx?direct=true&db=cmedm&AN=21233664&login.asp&site=ehost-live
95. Moore, Z., Cowman, S., Conroy, R. M. A Randomised Controlled Clinical Trial of Repositioning, Using the 30 tilt, for the Prevention of Pressure Ulcers. Journal of Clinical Nursing 20(17-18):2633-44, 2011.
96. Källman, U., Engström, M., Bergstrand, S., et al. The Effects of Different Lying Positions on Interface Pressure, Skin Temperature, and Tissue Blood Flow in Nursing Home Residents. Biological Research for Nursing 17(2):142-51, 2015.
97. Knox, D. M., Anderson, T. M., Anderson, P. S. Effects of Different Turn Intervals on Skin of Healthy Older Adults. Advances in Wound Care 7(1):48-52, 54-46, 1994.
98. Kosiak, M. Etiology and Pathology of Ischemic Ulcers. Archives of Physical Medicine and Rehabilitation 40(2):62-9, 1959.
99. Bliss, M. R. Pressure Sore Management and Prevention. In J. C. Brocklehurst, et al., eds. Textbook of Geriatric Medicine and Gerontology, 4th ed. London: Churchill Livingstone, 1992.
100. Yap, T. L., Kennerly, S. M., Simmons, M. R., et al. Multidimensional Team-Based Intervention Using Musical Cues to Reduce Odds of Facility-Acquired Pressure Ulcers in Long-Term Care: A Paired Randomized Intervention Study. Journal of the American Geriatrics Society 61(9):1552-9, 2013.
101. Defloor, T., De Bacquer, D., Grypdonck, M. H. The Effect of Various Combinations of Turning and Pressure Reducing Devices on the Incidence of Pressure Ulcers. International Journal of Nursing Studies 42(1):37-46, 2005.
102. Vanderwee, K., Grypdonck, M. H., Defloor, T. Effectiveness of an Alternating Pressure Air Mattress for the Prevention of Pressure Ulcers. Age and Ageing 34(3):261-7, 2005.
103. Mimura, M., Ohura, T., Takahashi, M., et al. Mechanism Leading to the Development of Pressure Ulcers Based on Shear Force and Pressures During a Bed Operation: Influence of Body Types, Body Positions, and Knee Positions. Wound Repair and Regeneration 17(6):789-96, 2009.
104. Vollman, K. M. Hemodynamic Instability: Is It Really a Barrier to Turning Critically Ill Patients? Critical Care Nurse 32(1):70-5, 2012.

105. Santamaria, N., Gerdtz, M., Sage, S., et al. A Randomised Controlled Trial of the Effectiveness of Soft Silicone Multi-layered Foam Dressings in the Prevention of Sacral and Heel Pressure Ulcers in Trauma and Critically Ill Patients: The Border Trial. International Wound Journal 12(3):302-8, 2015.
106. Bååth, C., Idvall, E., Gunningberg, L., et al. Pressure-reducing Interventions Among Persons with Pressure Ulcers: Results from the First Three National Pressure Ulcer Prevalence Surveys in Sweden. Journal of Evaluation in Clinical Practice 20(1):58-65, 2014.
107. Zernike, W. Preventing Heel Pressure Sores: A Comparison of Heel Pressure Relieving Devices. Journal of Clinical Nursing 3(6):375-80, 1994.
108. Call, E., Pedersen, J., Bill, B., et al. Enhancing Pressure Ulcer Prevention Using Wound Dressings: What Are the Modes of Action? International Wound Journal 12(4):408-13, 2015.
109. Cheneworth, C. C., Hagglund, K. H., Valmassoi, B., et al. Portrait of Practice: Healing Heel Ulcers. Advances in Wound Care 7(2):44-8, 1994.
110. Flemister, B. A Pilot Study of Interface Pressure with Heel Protectors Used for Pressure Reduction. Journal of ET Nursing 18(5):158-61, 1990.
111. Moody, B. L., Fanale, J. E., Thompson, M., et al. Impact of Staff Education on Pressure Sore Development in Elderly Hospitalized Patients. Archives of Internal Medicine 148(10):2241-3, 1988.

12

Pain Management and Wounds

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We gratefully acknowledge the contributions of Linda E. Dallam, MS, RN-BC, GNP-BC, CWCN-AP, for her work on previous editions of this chapter.

Objectives

After completing this chapter, you'll be able to:

- define and identify the components of wound-associated pain
- describe the similarities and differences of pain associated with various types of chronic wounds
- utilize validated patient tools for chronic wound-related pain
- assess the advantages and disadvantages of wound pain treatment modalities.

Etiology and Definitions of Pain

Pain has an element of blank;It cannot recollectWhen it began, or if there wereA day when it was not.

—Emily Dickinson

As clinicians, we have a tendency to identify various wound types as having *characteristic* pain of a specific type or amount. However, pain is what the patient states it is—not what we believe it to be. Our responsibility as clinicians is to assess the patient's pain accurately and treat it adequately, without judging the patient or doubting that the pain is as described. Pain is often more important to patients than it is to clinicians. Surveys indicate that pain is the most important parameter for many patients but is often only the third or fourth priority for clinicians.

There are several definitions of pain in the literature. Both the 1979 International Association for the Study of Pain (IASP) Subcommittee on Taxonomy[1] and the Agency for Healthcare Research and Quality (AHRQ, formerly the Agency for Healthcare Policy and Research, or AHCPR)[2] support a common definition of pain. They have defined pain as *an unpleasant sensory and emotional experience associated with actual or potential tissue damage or described in terms of such damage.*[1,2]

Another commonly used pain definition is that of McCaffery and colleagues,[3,4] who state that "pain is whatever the experiencing person says it is and exists whenever he (she) says it does."

The inability to communicate verbally or behaviorally does not negate the possibility the person is experiencing pain. This definition of pain encompasses the subjective component and acknowledges the patient as the best judge of his or her own pain experience. Experts in the field of pain have come to accept that the patient's self-reporting of pain, including its characteristics and intensity, encompasses the most reliable assessment. This belief that the patient in pain is his or her own best judge is also accepted as the

basis for pain assessment and management by such regulatory agencies as the Joint Commission, formerly known as the Joint Commission on Accreditation of Healthcare Organizations[5] as well as such professional organizations as the American Pain Society (APS).[6]

Practice Point

Pain is what the person says it is and exists whenever he or she says it does. The true etiology of pain isn't known. More research is needed to learn the true cause of the individual patient's pain.

It is important for clinicians to assess pain in every person with chronic wounds. A comprehensive Australian study documented chronic wound pain assessment behavior in 1,190 members of the Wounds Australia Organization. Nurses were more likely to assess wound-related pain at dressing change (61%) with two-thirds using a validated assessment tool. Podiatrists were less likely to assess pain usually only when they received patient-initiated complaints.[7]

Types of Pain

Pain can be nociceptive or neuropathic or have components of both as commonly experienced with wound-associated pain. Nociceptive pain can result from ongoing activation of primary afferent neurons by noxious stimuli, with a normally functioning nervous system. Neuropathic pain is initiated or caused by a primary lesion or dysfunction of the nervous system.[8]

The two types of nociceptive pain are somatic and visceral. Somatic pain arises from the bone, skin, muscle, or connective tissue. It's usually gnawing, aching, tender, or throbbing and well localized. Pressure ulcer pain is usually somatic in nature. Visceral pain arises from the internal organs such as the gut or from an obstruction of a hollow viscous organ, as occurs with a blockage of the small bowel. Visceral pain is poorly localized and is commonly described as cramping. Both types of nociceptive pain respond well to nonopioid and opioid pain medication.

The origin of neuropathic pain is from an abnormal processing of the sensory input by the peripheral or central nervous system (CNS). Taverner, Closs, and Briggs'[9] grounded theory study of 11 older adults with leg ulcer–associated pain described three phases:

- Phase 1 has predominantly acute nociceptive properties.
- Phase 2 persistent pain develops when the original pain is not managed adequately or the ulcer fails to heal and this windup phenomenon results in persistent pain with both nociceptive and neuropathic properties.
- Phase 3 occurs when the persistent pain becomes refractory to usual treatment.

The neuropathic component of the pain is typically described as having burning, stabbing, stinging, shooting, or electrical characteristics. Diabetic neurotrophic foot ulcer pain and the pain of shingles are examples of neuropathic pain.

A 2014 publication[10] of effective analgesic agents for neuropathic pain included RCTs, systematic reviews, and existing guidelines.

Treatments were listed as 1st to 4th line:

1. Gabapentinoids (pregabalin, gabapentin), tricyclic antidepressants (nortriptyline, amitriptyline), serotonin/noradrenalin reuptake inhibitors (duloxetine [Cymbalta], venlafaxine [Effexor])
2. Tramadol, controlled-release opioid analgesics (fentanyl, long-acting morphine)
3. Cannabinoids
4. Methadone, lower-evidence anticonvulsants and botulinum toxin

Neuropathic pain responds more readily to adjuvant agents, including antidepressants (e.g., tricyclic, serotonin–norepinephrine reuptake inhibitors, SSRI), or anticonvulsant therapy. Tricyclic antidepressants (TCA), such as amitriptyline and nortriptyline, are good choices because of their antinoradrenaline activity. Amitriptyline is a first-generation tricyclic agent with almost equal antinoradrenaline, antihistamine, antiserotonin, and antiadrenergic actions. Nortriptyline is a second-generation tricyclic that has higher antinoradrenaline activity at a lower dose, with fewer side effects such as double vision, dry mouth, and urinary retention. Desipramine has the same advantages as nortriptyline with less drowsiness. Duloxetine is an SSRI that has been approved for the treatment of painful peripheral diabetic neuropathy. The analgesic effect is likely related to augmentation of the inhibitory pain pathways in decreasing the perception of pain. If antidepressant agents are not tolerated or provide inadequate relief of neuropathic pain at reasonable dosages, then anticonvulsants, such as gabapentin and its derivative pregabalin, should be considered. Gabapentin has demonstrated a reduction in neuropathic pain.[11] Pregabalin has also proved to be useful in the treatment of neuropathic pain, with studies demonstrating a benefit in painful postherpetic neuropathy[12] and painful diabetic neuropathy.[13] Both gabapentin and pregabalin require dose adjustments in patients with renal disease.

Pain can also be acute (intermittent) or chronic (persistent). Acute pain has a distinct onset, with an obvious cause and short duration, and is usually associated with acute wounds, subsiding as healing takes place. Chronic pain can be from a chronic wound or other long-

term disease, such as cancer. If it persists for 3 months or more, chronic pain is usually associated with functional and psychological impairment. Chronic pain can fluctuate in character and intensity.

The American Geriatric Society (AGS)[14] supports the terminology of “persistent” pain rather than “chronic” pain to circumvent the negative stereotypes that have been associated with the word “chronic.” The AGS Clinical Practice Guideline, “The management of persistent pain in older persons,” states: “Unfortunately, for many elderly persons, chronic pain has become a label associated with negative images and stereotypes commonly associated with long-standing psychiatric problems, futility in treatment, malingering, or drug-seeking behavior. Persistent pain may foster a more positive attitude by patients and professionals for the many effective treatments that are available to help alleviate suffering”.[14]

Persistent and acute wound-associated pain can occur at the same time; similarly, nociceptive and neuropathic pain may occur simultaneously. Wound-associated pain is often a combination of nociceptive and neuropathic pain. It may be compounded by an inflammatory process that occurs from local tissue damage due to surgery, infection, trauma, or other inflammatory conditions. Inflammation and infection are characterized by redness, heat, and swelling that have been associated with an increased sensitivity to pain in and around the wound site.[15–17] Inflammation or infection-associated pain usually resolves when the condition that provoked the pain is controlled. Local tissue ischemia has also been implicated as a contributing factor to increased pain sensation in the acute phase of wounds.[15]

Regardless of the types and etiologies, pain can be debilitating and associated with sleep disturbance, poor appetite, functional decline, and/or psychosocial maladjustment. Patients suffering from pain reported decreased quality of life that extends beyond its physical component.

Practice Point

Reframing the phrase “the patient complains of pain” to “the patient reports pain” may help to foster a more positive and objective way for practitioners and caregivers to connect with the patient’s experience of pain. Use the term *persistent pain* rather than *chronic pain*.

The Persistent (Chronic) Pain Experience

Krasner[18–20] has conceptualized pain in chronic wounds as the chronic wound pain experience. Within this model, pain is divided into three categories: noncyclic, cyclic, and chronic pain. *Noncyclic* or *incident* pain is defined as a single episode of pain that might occur, for example, after wound débridement. *Cyclic* or *episodic* pain recurs as the result of repeated treatments, such as dressing changes, débridement, or turning and repositioning. *Chronic* or *continuous* pain is persistent and occurs without manipulation of the patient or the wound. For example, the patient may feel that the wound is throbbing even when he or she is lying still in bed and with no treatment occurring at the local wound site.

Practice Point

Interventions for Noncyclic Wound Pain

- Identify and develop a pain treatment plan for potentially painful procedures.
- Administer topical or local anesthetics.
- Consider an operating room procedure under general anesthesia rather than bedside débridement for large, deep ulcers.
- Administer opioids and/or nonsteroidal anti-inflammatory drugs before and after procedures.
- Assess and reassess for pain before, during, and after procedures.
- Avoid using wet-to-dry dressings that can cause pain and trauma upon removal.

- Consider alternatives to surgical/sharp débridement, such as transparent dressings, hydrogels, hydrocolloids, hypertonic saline solutions, or enzymatic agents.[17]

Practice Point

Interventions for Cyclic Wound Pain

- Perform interventions at a time of day when the patient is less fatigued.
- Provide analgesia 30 to 60 minutes before dressing change.
- Assess the patient for pain before, during, and after dressing changes.
- Provide analgesia 30 to 60 minutes before whirlpool.
- If the patient's dressing has dried out, thoroughly soak the dressing—especially the edges—prior to removal.
- Observe the wound for signs of local infection.
- Gently and thoroughly cleanse or irrigate the wound to remove debris and reduce the bacterial bioburden, which can cause contaminated wounds to become critically colonized or infected. Infection will increase the inflammation and pain at the wound site.
- Avoid using cytotoxic topical agents.
- Avoid aggressive packing. (Fluff; do not stuff!)
- Avoid drying out the wound bed and wound edges.
- Protect the periwound skin with sealants, ointments, or moisture barriers.
- Minimize the number of daily dressing changes.
- Select pain-reducing dressings that include moisture balance for healable wounds, consider soft silicone adhesives, and avoid aggressive adhesives.

- Avoid using regular/aggressive tape on fragile skin—consider silicone adhesive tape.
- Splint or immobilize the wounded area as needed.
- Utilize pressure-reducing devices in bed or when seated in a chair.
- Provide analgesia as needed to allow positioning of patient.
- Avoid trauma (shearing and tear injuries) to fragile skin when transferring, positioning, or holding a patient.

Practice Point

Interventions for Persistent (Chronic) Wound Pain

- Use all of the interventions listed for noncyclic and cyclic wound pain.
- Control edema.
- Control infection.
- Monitor wound pain while the patient is at rest (at times when no dressing change is taking place).
- Control pain to facilitate healing and positioning.
- Provide regularly scheduled analgesia, including opioids, patient-controlled analgesia, and topical preparations such as lidocaine gel 2%, depending on the severity of pain.
- Attend to non–wound-associated pain from:
    - Comorbid pain syndromes such as contractures and neuropathic pain associated with diabetes/other neuropathies
    - Iatrogenic device insertions, such as central lines, venous puncture sites, catheters, feeding tubes, blood gas drawing, or other equipment or procedures

- Address the emotional component of the pain or the patient's suffering:
    - What does the wound represent to the patient?
    - What does pain mean? Is it associated with loss of function?
    - Has the wound altered the patient's body image?
    - Did unrelieved pain alter the patient's mental status or behavior?

Pain and Wound Types

The type of pain a patient experiences depends largely on the type of wound present. Pain can occur in patients with acute and chronic wounds and can be related or unrelated to the wound or its cause. Clinicians should determine whether pain is generalized, regionalized, or related directly to the wound bed. Regional pain often relates to the wound cause. Localized wound pain may relate to local wound manipulation, treatment modalities, or infection.[21] Gardner and Frantz[21] identified increased wound-associated pain as a potential symptom of infection. This section discusses various types of wounds and the types of pain that accompany them.

Pressure Injury Pain

Pain at the site of a pressure injury is supported by pressure injury experts and anecdotal reports by clinicians, although few studies concerning pressure injury pain have been published. At its first conference in 1989, the National Pressure Injury Advisory Panel (NPIAP) stated that "pressure ulcers are serious wounds that cause considerable pain, suffering, disability, and even death."[22] van Rijswijk and Braden[23] reevaluated the AHCPR Treatment of Pressure Ulcer guidelines in light of studies published after the guidelines were released in 1994[24] and reaffirmed the panel's first recommendation about assessing pressure injury patients for pain. Based on additional evidence from studies supporting pain reduction with the use of moisture-retentive dressings, however, van Rijswijk and Braden[23] proposed that the 1994 AHCPR recommendations concerning pain and pressure injuries be rewritten.

The etiology of pain in patients with pressure ulcers is often unknown. Pieper[25] quotes the work of Rook[26] and suggests that the common sources of pressure injury pain are from the "release of noxious chemicals from damaged tissue, erosion of tissue planes with destruction of nerve terminals, regeneration of nociceptive nerve terminals, infection, dressing changes, and débridement." In stage 3 or 4 pressure injuries, this pain may come from ischemic necrosis of

the tissue triggered by a deep tissue injury or shear forces. Superficial stage 2 ulcers may be associated with skin surface pain from moisture or friction.

According to a study by Szors and Bourguignon,[27] pressure ulcer pain depends not only on the stage of the injury but also on whether a dressing change is taking place at the time the assessment is made. The majority of patients in their study (88%) reported pressure injury pain with dressing changes; an almost equivalent number of patients (84%) had persistent pain at rest. Patients rated the pain from sore to excruciating. Seventy-five percent rated their pain as mild, discomforting, or distressing; 18% rated their pain as horrible or excruciating. Clinicians need to ensure adequate pain control for patients with persistent pain with long-acting pain and breakthrough medication; they must also time the breakthrough medication so that it is effective against the pain experienced at dressing change. In addition, appropriate cleansing and débridement methods and suitable dressings need to be chosen that will minimize wound surface pain and trauma at the time of removal and reapplication.

In 2009, Pieper and colleagues[28] published an NPIAP position paper based on a systematic review of the pressure injury pain literature of which 15 studies met their inclusion criteria. Four studies were on topical medication treatment and the others on various other pain treatments, assessment, and the pain experience. Their findings included choosing dressings that minimize pain. They were unable to find studies regarding nutrition and pressure injury pain but did discuss the potential of nausea and decreased appetite for patients with pressure injuries experiencing pain. These authors were also able to identify gaps in the research literature for special groups such as neonates, children, bariatric, and end-of-life patients. In 2010, Langemo and Black published an NPIAP white paper that includes recommendations for assessing, preventing, and treating pressure ulcer pain in the population of 300 million individuals who receive palliative or end-of-life care.[29]

Arterial and Lower Limb Ulcer Pain (Including Diabetic Foot Ulcers)

Pain associated with peripheral vascular disease can be due to intermittent claudication or to rest pain with advanced disease that may be more prominent at night with leg elevation. Intermittent claudication pain results from physical exertion or exercise-induced ischemia and has been described as cramping, burning, or aching. The blood flow with increasing exertion becomes inadequate to meet the needs of tissues. However, only 10% to 35% of patients report classic claudication and 40% to 50% atypical leg pain during the early stages of the disease.[30]

Patients can employ several tactics to relieve pain. The most important are to stop smoking, start gradual and regular exercises, lose weight, and have vascular risk factors treated.

Nocturnal pain may have the same symptoms but usually precedes the occurrence of rest pain. Rest pain occurs—even without activity—when blood flow is inadequate to meet the needs of tissues in the extremities. These types of pain are described as a sensation of burning or numbness aggravated by leg elevation where gravity no longer can facilitate local blood flow. The pain is constant and intense and isn't easily relieved by pain medications. Pain can sometimes be alleviated by stopping the activity or exercise and placing the legs in a dangling or dependent position (head of bed on blocks) to promote gravity-assisted blood flow. Some patients find relief with gentle massage, while other wear socks to help maintain warmth. A recent review by Newbern[31] cautions clinicians that there is a lack of a validated pain and Q of L assessment tool for diabetic foot ulcers or chronic lower-extremity wounds, with the result that single question pain assessments may undertreat pain especially the psychosocial aspects and Q of L-related symptoms. The authors also caution that "symptom clusters including pain, fatigue, exudate, inflammation and edema profoundly affect patient Q of L and may increase the risks associated with delayed healing".[31]

Venous Ulcer Pain

Venous ulcer pain can have several possible sources:

- Edema due to extravasated fluid from the capillaries

- Inflammation of woody fibrosis: acute or subacute lipodermatosclerosis
- Bacterial damage
- Superficial local infection/increased bacterial burden (NERDS©)[32,33]
- Deep and surrounding skin infection (STONES©)[32,33]
- Inflammation of the veins: superficial and deep phlebitis

The range of venous ulcer pain is extensive; the patient may report mildly annoying pain, a dull ache, or sharp, deep muscle pain. Pain is more intense at the end of the day secondary to edema from the legs being in a dependent position. This dependent edema is often aggravated by standing, sitting, or crossing the legs. The pathophysiology of venous disease is related to reduction or occlusion of blood returning to the heart. Incompetent superficial, perforating, or deep veins can cause pooling of fluid in the legs leading to pitting edema and resultant pain. To minimize pain, patients should be instructed to elevate the legs (as high as the heart on the armrest of a two-seater couch) when sitting and encouraged to wear support stockings. Stocking selection is based on accurate individualized measurement, and their effectiveness relies on putting them on before the legs are placed on the floor in the morning. Other clinical management goals that help to minimize venous-related edema include the avoidance of prolonged sitting, weight reduction, and smoking cessation.

Superficial phlebitis is associated localized tenderness and pain over the long and short saphenous veins that are aggravated by palpation. The involved area is often red and follows the outline of the vein. The pain can often be treated with topical nonsteroidal anti-inflammatory drug (NSAID) creams or gels. Thrombus formation in the deep veins (deep thrombophlebitis) can lead to diffuse pain and deep leg swelling that may mimic a deep and surrounding infection or acute lipodermatosclerosis. When the deep thrombophlebitis extends above the knee, there is an increased incidence of clots being associated with pulmonary emboli.

Increased bacterial burden in the superficial wound bed (local infection) can lead to delayed healing and localized pain. Clinicians

should look for three or more NERDS[32,33] signs: **n**onhealing wounds; increased **e**xudate; **r**ed, friable granulation tissue; new **d**ebris or slough on the surface; and an unpleasant **s**mell or odor. (For more information about NERDS and STONES, see Chapter 7, Wound Bioburden and Infection.)

When venous disease has been present for a long period of time, the veins become leaky with fibrin extravasation into the dermis (woody fibrosis). In addition, red blood cells can leak into the tissue, causing hyperpigmentation due to a combination of hemosiderin and melanin. The woody fibrosis does not go away at the end of the day, and patients can have acute and chronic inflammatory changes within the woody fibrosis, leading to acute (often severe pain) and chronic lipodermatosclerosis pain (subacute, chronic). In a recent study by Woo and Marin,[34] mixed arteriovenous leg ulcers were associated with lower health-related quality of life, greater mobility impairments, and more deficits in self-care and usual activities. Participants in the study rated pain as one of the most common symptoms with two-thirds of them reporting moderate/severe levels of pain.

Neuropathic Ulcer Pain

Neuropathy is the most common complication of diabetes. The amount of pain present depends on the severity of the neuropathy. Unlike stimulus-dependent nociceptive pain, neuropathic pain is spontaneous. The patient may state that the pain interferes with his or her entire life—especially the ability to sleep. The affected extremity may feel like it's asleep ("a block of wood") or have the "pins and needles" pain that occurs after a part of the body has "fallen asleep" and starts to wake up. The quality of pain can be burning, stinging, stabbing, or shooting and may include increased skin sensitivity to nonnoxious stimuli (allodynia) and itching. True pain relief is accomplished primarily with pharmacologic intervention. All pain needs to be assessed adequately to ascertain the most effective treatment modality. If a patient reports excessive pain in a neuropathic limb that hasn't had pain before, an infection or acute Charcot joint changes may be developing.

Patients with diabetes lose protective sensation after 10 to 15 years (sooner with poor blood glucose control). This loss of protective sensation allows these individuals to undergo sharp surgical débridement without nociceptive pain, although they may have referred pain in the leg or foot. If persistent nociceptive pain develops in a neuropathic limb, it usually means there's disruption of the deeper structures. In a person with a foot ulcer, clinicians should check for underlying osteomyelitis. If a patient has a tender, swollen foot without ulceration and an increase in skin surface temperature, there's a strong possibility of a Charcot foot. Occasionally, a patient may have both osteomyelitis and a Charcot foot.

Practice Point

Determining whether pain in a person with diabetes is the result of neuropathy or is associated with peripheral vascular disease or infection is extremely important because patients with diabetes have a high incidence of peripheral vascular disease. In addition, pain in a painless foot usually indicates disruption of the deeper structures and a strong possibility of associated osteomyelitis, Charcot foot, or even both conditions coexisting.

Pain Associated with Burns and Skin Graft Donor Sites

A recent article[35] on pediatric burn pain highlights the COMFORT behavioral scale for assessing the degree of pain. Each of the six categories (alertness, calmness–agitation, crying, physical movement, muscle tone, facial tension) has anchors on a 1 to 5 scale. Higher numbers indicate more severe pain. Values can range from 6 to 30 (6 to 13 mild pain, 14 to 20 moderate pain, 21 to 30 severe pain).

This scale helps to identify often untreated burn-associated pain that may have serious medical and psychiatric consequences. Traditional pain strategies may need to be supplemented with regional anesthesia and novel technologies including virtual reality. There are three components in burn-related pain that may require supplementation including background pain (consider benzodiazepines for sedation), procedural pain (virtual reality, multimodal distraction), and perioperative pain (nerve blocks).[36]

Split-thickness skin grafts are often utilized in burn patients or to close chronic stalled wounds. Novel RCT proven approaches to the skin graft–associated pain include subcutaneous local anesthetic infusion and injections, ice application, topical lidocaine or bupivacaine, and dressings with hydrocolloids or polyurethane with fibrin sealants.[37]

Pain Associated with Hidradenitis Suppurativa

Hidradenitis suppurativa is an inflammatory condition of the flexural hair follicles (axilla, under female breasts, groin, and perianal regions) and not primarily in the apocrine glands as previously believed. It can affect patient's quality of life more than any other dermatological conditions due to the pain and odiferous discharge from inflamed lesions.[38]

Pain management can start with topical treatment of washing the involved area with topical chlorhexidine containing preparations without alcohol (mouthwashes used off-label) followed by topical acne preparations containing benzoyl peroxide and clindamycin combinations (*personal communication—RG Sibbald*). Oral antibiotics, often tetracyclines (doxycycline is preferred), can treat deeper nodules or papules with an anti-inflammatory dose of 40 to 100 mg or an antistaphylococcal dose of 100 mg bid. Acetaminophen (often 1 g tid) or NSAIDs are often used for first-line pain control with oral tricyclics, gabapentin/pregabalin, duloxetine/venlafaxine, or opioids used in resistant cases; topical fluid lock superabsorptive dressings (use hygiene pads), ice packs, and topical NSAID or Xylocaine gels may also be useful.[39]

Understanding Wound Pain

Most of our understanding of wound pain comes from literature about other diseases.[40] Clinicians are increasingly acknowledging that pain is a major issue for patients suffering from many different types of wounds.[40] Several consensus statements and other documents regarding wound pain during dressing changes are available to help clinicians manage this type of pain properly.

Practice Point

Pain: What We Know, What We Don't Know

McCaffery and Robinson[4] reported on nurses' self-evaluation of their knowledge about pain.

- Observable changes in vital signs must be relied upon to verify a patient's report of severe pain: False (answered correctly by 88.4%).
- Pain intensity should be rated by the clinician, not the patient: False (answered correctly by 99.1%).
- A patient may sleep in spite of moderate or severe pain: True (answered correctly by 90.6%).
- Intramuscular (IM) meperidine is the drug of choice for prolonged pain: False (answered correctly by 85.6%).
- Analgesics for chronic pain are more effective when administered as needed rather than around the clock: False (answered correctly by 92.7%).
- If the patient can be distracted from the pain, the patient has less pain than he or she reports: False (answered correctly by 94.7%).
- The patient in pain should be encouraged to endure as much pain as possible before resorting to a pain relief

measure: False (answered correctly by 98.4%).

- Respiratory depression (<7 breaths/min) probably occurs in at least 10% of patients who receive one or more doses of an opioid for relief of pain: False (answered correctly by 60.5%; clinicians tend to exaggerate the risk of respiratory depression with opioid use; according to McCaffery and Robinson, the risk is <1%).
- Vicodin (hydrocodone 5 mg and acetaminophen 500 mg) is approximately equal to the analgesia of one-half of a dose of meperidine 75 mg IM: False (correctly answered by 48.3%).
- If a patient's pain is relieved by a placebo, the pain isn't real: False (answered correctly by 86.1%).
- Beyond a certain dose, increasing the dosage of an opioid such as morphine won't increase pain relief: False (answered correctly by 57.2%).
- Research shows that promethazine reliably potentiates opioid analgesics: False (correctly answered by 35.1%).
- When opioids are used for pain relief under the following circumstances, what percentage of patients is likely to develop opioid addiction?
    - Patients who receive opioids for 1 to 3 days: Answer is less than 1% (correctly answered by 82.8%).
    - Patients who receive opioids for 3 to 6 months: Answer is less than 1% (correctly answered by 26.7%).

International Guidelines

The European Wound Management Association (EWMA-2002)[41] has developed a position document on wound pain titled “Pain at wound dressing changes.” The document is subdivided into three sections:

- Understanding wound pain and trauma from an international perspective[42]
- The theory of pain[43]
- Pain at wound dressing changes: a guide to management[44]

In the first section of the document, Moffat and colleagues[41,45] surveyed 3,918 healthcare professionals from the United States and 10 countries in Western and Eastern Europe. The survey respondents indicated that pain prevention was the second highest ranking consideration at dressing change, with trauma prevention being first.[41,45] Pain from leg ulcers was ranked as the most severe pain compared with other wound types, and dressing removal caused the greatest pain.[26]

A copy of this EWMA position document[41,45–47] can be found on the Internet and is available in Dutch, English, French, German, Italian, and Spanish (Table 12-1).

Table 12-1 EWMA Suggestions for Preparing the Wound Pain Environment[41,45–47]

Prepare, Plan, Prevent

- Choose an appropriate nonstressful environment, close windows, turn off mobile phones, etc.
- Explain to the patient in simple terms what will be done and the method used.
- Assess the need for skilled or unskilled assistance, such as help with simple hand holding.
- Be thoughtful in positioning the patient to minimize discomfort and avoid unnecessary contact or exposure.
- Avoid prolonged exposure of the wound (e.g., waiting for specialist advice).
- Avoid any unnecessary stimulus to the wound, and handle wounds gently, being aware that any slight touch can cause pain.
- Involve the patient throughout; frequent verbal checks and use of pain tools offer real-time feedback.
- Consider preventive analgesia.

Another international consensus statement was developed by the World Union of Wound Healing Societies (WUWHS)[42]. This document, entitled “Principles of best practice: Minimizing pain at wound dressing related procedures,” outlines common pain management challenges, myths, and misunderstandings that are useful for improved clinical practice during wound dressing changes (Table 12-2). This document also includes helpful suggestions for care planning and treatment interventions (Table 12-3).

Table 12-2 Dispelling Myths About Wound Pain

In its international consensus statement,[42] WUWHS outlines several myths related to wound pain.

- **Myth:** Wet-to-dry dressing is still the gold standard for wound care.
- **Fact:** Adherent gauze can disrupt delicate healing tissue and provoke severe pain.
- **Myth:** Transparent films are the best dressing for treating and reducing the pain of skin tears and other minor acute wounds.
- **Fact:** The misuse of transparent films is a common cause of skin tears.
- **Myth:** Using paper tape is the least painful way to secure a dressing.
- **Fact:** Heightened nerve sensation in a wide area around a wound can make adhesive tape painful to remove.
- **Myth:** Pulling a dressing off faster rather than slower reduces pain at dressing changes.
- **Fact:** This method has the potential to inflict tissue damage and traumatic pain.
- **Myth:** Using a skin sealant on periwound skin reduces the risk of pain and trauma.
- **Fact:** Skin sealants only create a thin topical layer and do not protect deeper dermal layers.
- **Myth:** People with diabetic foot wounds do not experience pain.
- **Fact:** There may be some loss of peripheral nerve sensation, but sensitivity in the area can be heightened.
- **Myth:** Pain comes from the wound. The surrounding tissue nerves play little role.
- **Fact:** Spinal cord responses to incoming pain signals can give rise to abnormal sensitivity in the surrounding area (allodynia).
- **Myth:** The only way to treat wound pain is by oral analgesic, 30 to 60 minutes before dressing changes.
- **Fact:** An oral analgesic can give some relief but should not be seen as a single solution. A full pain assessment must be used to evaluate and fine-tune any prescribed therapy.

Table 12-3 WUWHS Procedural Wound Pain Interventions

The following planning and treatment suggestions from WUWHS[42] can be helpful in caring for patients with wound pain:

- Be aware of the current status of pain
- Know pain triggers, and avoid them where possible
- Know and use pain reducers (when possible and not contraindicated)
- Avoid unnecessary manipulation of the wound
- Explore with the patient simple distraction techniques, such as counting up and down, focusing on the breath entering and leaving the lungs, or listening to music
- Reconsider management choices if pain becomes intolerable, and document as an adverse event
- Observe the wound and surrounding skin for evidence of infection, necrosis, or maceration
- Consider the temperature of the product before applying it to the wound
- Avoid excessive pressure from a dressing, bandage, or tape
- Follow the manufacturer's instructions when using a dressing or technology
- After the procedure, assess comfort of intervention and/or dressing/bandages applied

Ongoing evaluation and modification of the management plan and treatment intervention is essential as wounds change over time. More advanced nonpharmacologic techniques that require specialist training or skilled personnel, such as the use of hypnosis or therapeutic touch, may be considered.

Lastly, a third international document (not a consensus statement) addresses the management of pain associated with burns. This document is titled "The management of pain associated with dressing

changes in patients with burns" and can be found in the electronic wound care journal, *World Wide Wounds*.[43]

Pain Research: Pressure Injuries

Nursing Management

Nursing management of patients with pressure injuries sometimes does not adequately address the component of pain management. Hollingworth[40] determined that nurses' assessment, management, and documentation of pain after completing a wound dressing change were inadequate. Likewise, in a qualitative study that examined the reflections of 42 general and advanced practice nurses, Krasner[44] identified three distinct patterns nurses used to address pressure injury pain in their patients—nursing expertly, denying the pain, and confronting the challenge of pain:

1. Nursing expertly
    - Reading the pain
    - Attending to the pain
    - Acknowledging and empathizing with the patient
2. Denying the pain
    - Assuming that it doesn't exist
    - Not hearing the cries
    - Avoiding failure
3. Confronting the challenge of pain
    - Coping with frustration
    - Being with the patient[44]

Krasner[19,20,44] suggested that clinicians use this information to provide more patient-centered sensitive care for patients with pressure injury pain.

The Patient's Pain Experience

Few studies (four quantitative and five qualitative) have been published concerning the pain experience of patients with pressure injuries. Variability in pain perceptions can be influenced by many contextual and psychological (patient-centered) factors. Woo[48] examined 96 patients with chronic wounds to determine whether

anxiety or anticipatory pain played a role in the intensity of pain experienced at dressing changes. Dr. Woo uncovered a direct relationship between anxiety related to impending pain that subsequently, intensified the experienced pain. With heightened anxiety, environmental and somatic signals are brought to the patient's attention, sharpening the degree of sensory receptivity and reducing pain tolerance. Woo documented that certain individuals who are insecure in their relationship with others were susceptible to experiencing anxiety and intensified pain.[48]

Dominic Upton and colleagues[49] linked stress and pain at dressing change to delayed healing of chronic wounds. The same group documented stress assessment tools that may capture an additional component of experienced pain that is often missing when only pain is assessed.[50]

The first study to quantify pain by pressure injury stage was completed by Dallam and colleagues,[17] who documented the perceived intensity and patterns of pressure injury pain in hospitalized patients. The study population was diverse, with 66% being white (non-Hispanic) and the remainder being black (non-Hispanic), Hispanic, or Asian. Of the 132 patients enrolled in the study, 44 (33.3%) were respondents and 88 (66.7%) were nonrespondents because they couldn't communicate responses to the instruments (language and other cognitive barriers). Two different scales were used to measure pain intensity: the Visual Analog Scale (VAS) and the FACES Pain Rating Scale (FPRS). The authors identified a high degree of agreement between the two pain scales. They also noted that the FPRS was easier to use for patients who were cognitively impaired or for whom English was their second language.

The major findings of this important study include the following[17]:

- The majority of patients with pressure injuries had injury-related pain (68% of respondents reported some type of pain).
- Most patients didn't receive analgesics for pain relief; only 2% (n = 3) of patients in this population were given analgesics for pressure injury pain within 4 hours of the pain measurement.

- Patients who couldn't express pain or respond to pain scales may still have had pain.
- Patients with deeper pressure injury stages (stages 3 and 4) had more pain.

Some procedures, such as surgical débridement or wet-to-dry dressing changes, may increase pain. While the study didn't identify the interventions that might be most effective in controlling pain, patients whose beds had static air mattresses rather than regular hospital bed mattresses and those whose wounds were dressed with hydrocolloid dressings had significantly less pain.[17] The study also demonstrated that patients are able to differentiate between injury site pain, generalized pain, and other local pain sites such as IV and catheter sites.[17] Some cognitively impaired patients were able to indicate the presence of pain and respond to pain intensity scales.

Both Dallam et al.[17] and Szors and Bourguignon[27] discovered that many patients suffer with untreated or undertreated pressure injury–associated pain. Dallam and colleagues[17] determined that only 2% of patients with pressure injury pain received analgesia. Four years later, Szors and Bourguignon's[27] evaluation documented little improvement in the administration of pain-relieving medication: only 6% of patients with pressure injury pain had analgesics prescribed to address their pain.

Both studies reflect the need for clinicians to realize the potential for pain from pressure injuries. Because only 44 of the 132 patients with pressure injury pain could respond to pain scales, Dallam and colleagues[17] recommend that pressure injury pain should be suspected even when the patient can't report pain. Both studies recommend further research to identify interventions that can relieve pressure injury pain and the associated suffering.

Franks and Collier[51] conducted a study in the United Kingdom in which they compared home care patients with (n = 75) and without (n = 100) pressure injuries. Interestingly, they documented that patients with pressure injuries had less pain than did those who did not have pressure injuries. The authors speculated that perhaps pressure injury pain might not be the problem, as previously presumed, or that pain

control was somehow more effective for patients receiving home care. An alternative explanation is that the home care patients with pressure injuries did not have the same comorbid conditions as previously reported in hospital populations or that the comparator conditions evaluated in home care may have had greater pain (e.g., infected wounds).

In a quantitative pain study of 128 chronic wound patients, Ayello and colleagues[52] found that more than half of the patients with venous ulcers had pain (54%), almost one-third with diabetic neurotropic or neuroischemic ulcers had pain (30%), and one-quarter of those with pressure ulcers (25%) had pain.

Langemo and colleagues[53] published a qualitative phenomenological study about pain in pressure injury patients. They interviewed eight adults, half with active pressure injuries at the time of the study and the other half with healed pressure injuries. Seven themes were identified:

1. the perceived etiology of the pressure injury
2. life impact and changes
3. psychospiritual impact
4. extreme painfulness associated with the pressure injury
5. the need for knowledge and understanding
6. the need for and stress related to numerous treatments
7. the grieving process.

The fourth theme—extreme pain—was subdivided into three categories: intensity of pain, duration of pain, and analgesic use. Patients commonly referred to the intensity of pain from pressure ulcers with descriptors such as “it burned,” “feeling like being stabbed,” “sitting on a bunch of needles,” or “stinging.” Some examples of statements by actual study respondents include a woman with a stage 2 pressure injury who said, “I felt like somebody was getting a knife and really digging in there good and hard.” In the words of another male respondent, “They (pressure injuries) are very painful because no matter what way you put your bottom, it hurts”.[53]

Respondents also commented on the duration of the pain, with statements such as “the majority of the time, even when I was lying down, it hurt.” Pain continued to be a problem even after the pressure injury had healed. As one respondent stated, “Every now and again, it still hurts. But there is nothing there. This time there is nothing really there.” The fear of addiction resulting from analgesic use was expressed by some respondents. One respondent with a stage 4 pressure injury on the buttock commented, “I was constantly in pain and was taking morphine and other types of painkillers to try and ease the pain.”

Another qualitative study reported about the pain of 10 pressure injury patients.[54] Although Rastine had identified 22 themes, lack of communication and painful treatment interventions were the two most common complaints.[51] Some patients related accounts of communication failures that contributed to stress, tension, and anxiety.[54]

The European Pressure Ulcer Advisory Panel (EPUAP) funded a phenomenological study by Hopkins et al.[55] who identified endless pain as one of the three main themes in older people living with pressure injuries. The eight patients in this study were all over age 65 and had stage 3 or 4 pressure injuries for more than 1 month. None had spinal cord injuries, as suggested by Langemo and colleagues[53] for future qualitative pressure injury pain studies. The four subthemes of endless pain were constant pain presence, keeping still, equipment pain, and treatment pain. For some patients, keeping still reduced their pain: “I don’t dare move because everything then gets worse. I lie very still.” For others, pain was exacerbated by pressure-relieving equipment as well as dressing changes. All but one of the patients described their endless pain in a graphic way. “You put a bit of weight on your heel and (it) feels as though it’s burst open”.[53]

Chronic wound studies[56,57] and the studies cited in this chapter emphasize the importance of adequate pain assessment and treatment.

Pain Assessment

Despite the APS's identification of pain as "the fifth vital sign",[6] it isn't always included in the assessment of a patient's pressure ulcer. Dallam and colleagues[17] urged that pain be added to the assessment of pressure ulcers and that a patient's pain status be assessed during dressing changes as well as when the patient is at rest. They also cautioned clinicians to remember that the absence of a response or an expression of pain doesn't mean that the patient doesn't have pain. Despite research about the pain experience,[17,53,54,56–58] assessment of pain in persons with pressure injuries continues to be underreported.[52] Documentation of pain assessment may vary by chronic wound type, as patients with venous ulcers (63%) and diabetic foot ulcers (53%) in one study were more likely to have their pain assessment recorded compared with those with pressure ulcers (45%).[52]

Two assessment guides include pain as part of pressure ulcer assessment. The AHCPR-1994[24] treatment guidelines include an example of a sample pressure injury pain assessment guide in which there is a place to check either yes or no regarding the presence of pain. Ayello's[59,60] ASSESSMENT mnemonic asks the clinician to quantify the patient's pain experience, including the presence of pain, when the pain occurs (e.g., is it episodic or constant), and if the patient is receiving measures for pain relief. The caregiver checks one of the following boxes under T = tenderness to touch or pain:

- No pain
- Pain present on touch, anytime
- Pain only when performing injury care[59,60]

The mnemonic PQRST, which outlines the specific questions to ask the patient, is another useful tool for assessing a patient's pain[17] (Table 12-4).

Table 12-4 Essential Pain Assessment Elements

Use the PQRST mnemonic (shown below) to assess your patient's pain.

P = Palliative/provocative factors

- What makes the pain worse?
- What makes it better?

Q = Quality of pain

- What kind of pain are you experiencing?
- Would you describe it as:
  - gnawing, aching, tender, throbbing (nociceptive)?
  - burning, stinging, shooting, stabbing (neuropathic)?
  - or any combination thereof?
  - Do you have other symptoms with the pain, such as fever, chills, nausea, or vomiting?

R = Region and radiation of pain

- Where is the pain?
- Does the pain travel or remain in the same spot?

S = Severity of pain

- Would you describe your pain as none, mild, moderate, severe, or excruciating?
- Rate your pain on a scale from 0 to 10, with 0 representing "no pain" and 10 being "the worst imaginable pain."
- How would you rate the pain intensity at its worst, best, and now?

T = Temporal aspects of pain

- Is the pain better or worse at any particular time of the day or night?
- When does it start or when does it stop?
- Is it intermittent or constant, or does it occur only when you're moving?

A complete and thorough pain assessment enables the clinician to develop an effective pain treatment regimen and evaluate its effectiveness (Table 12-5). The American Society for Pain Management Nursing offers a position statement and clinical practice recommendations for pain assessment in specific patient populations—groups that clinicians may not always identify as needing pain assessment. These groups include patients with advanced dementia, infants and preverbal toddlers, and intubated and/or unconscious patients.[61] The Hartford Institute for Geriatric Nursing has produced a series on pain assessment called “Try This”.[62] These one-page (front and back) documents provide a succinct summary that covers the important points on pain assessment in older adults and in patients with dementia.[62]

Table 12-5 Additional Pain Assessment Elements

Include the following additional elements in your initial assessment plan and treatment:

- Detailed history consisting of:
  - medication usage
  - treatment history
  - previous surgeries and injuries
  - impact on quality of life and activities of daily living.
- Physical examination, emphasizing the body system involved in the pain complaint (e.g., the musculoskeletal or neurologic system)
- Psychosocial assessment, including family history of depression or chronic pain
- Appropriate diagnostic workup to determine the cause of pain and to rule out any contributing, treatable causes

A thorough pain assessment enables the clinician to develop an effective pain treatment regimen and evaluate its effectiveness.

Practice Point

Pain is the fifth vital sign.

Pain Intensity Scales

Pain intensity scales use a simple verbal, visual, or numeric measure to help determine how much pain the patient is experiencing. The gold standard for assessing pain intensity is self-report and the utilization of standard pain intensity instruments.[63,64] Pain intensity scales are one-dimensional, quantitative measures designed to measure the sensory aspect of a patient's pain and to obtain a more objective approximation of pain by minimizing inaccuracies.[64]

The use of pain intensity scales to quantify pain levels and determine patients' responses to pain treatments has been mandated by the Joint Commission for use in all hospitals.[5] Two of the most widely accepted and utilized pain assessment scales are the Numeric Pain Intensity Scale and the FACES Pain Rating Scale.[65] Another commonly used scale is the Visual Analog Scale, which consists of a 10-cm line that has no numbers on it. At one end is the term "no pain," and at the other end is the phrase "pain as bad as it could possibly be".[2]

Numeric Pain Intensity Scale (Numeric [Pain] Rating Scale)

The Numeric Pain Intensity Scale is considered the gold standard for pain assessment for adults and children over age 7.[2,65] This scale is a 10-cm line with the words “no pain” at one end, “worst possible pain” at the other end, and the numbers 0 to 10 running from one end of the scale to the other (Fig. 12-1). The patient is asked to select the number on the scale that represents the level of pain he or she is experiencing. Zero indicates no pain, 5 indicates moderate pain, and 10 indicates the worst possible pain.[2] Patients can often relate these anchors to 10 being slamming the car door on your thumb and 5 being a bee sting (personal communication Gary Sibbald). The Numeric Pain Rating Scale is sometimes presented verbally[2,65]; however, visual presentation may help to standardize the process of pain assessment and assist hearing-impaired patients. In addition, the scale has been translated into many languages.[8]

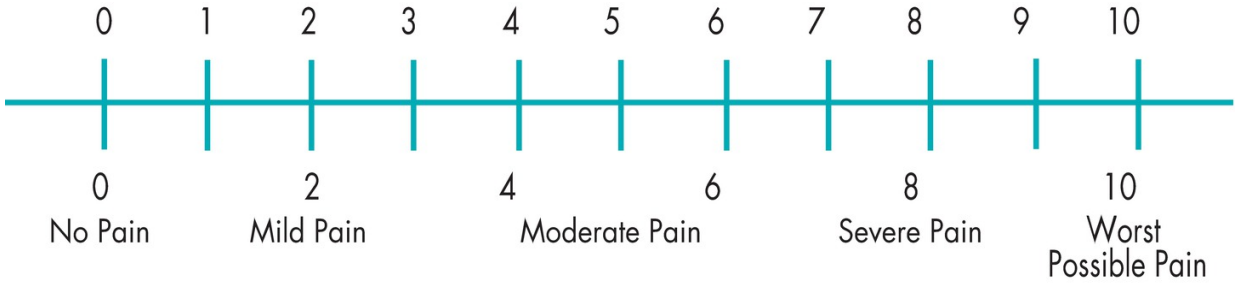

Figure 12-1. Numeric pain intensity scale. The Numeric Pain Intensity Scale is considered the gold standard for pain assessment and may be used for adults and children over age 7.

The Numeric Pain Rating Scale aids in the adequate assessment and treatment of pain. It also helps clinicians choose the appropriate classification of pain medication recommendations based on any

given patient response.[22,65,66] The scale can also help determine whether the patient has a response to the interventions if the numbers show a downward trend on repeated assessments.

FACES Pain Rating Scale

The FPRS[67] consists of six faces that range from a happy, smiling face (no pain) to a crying, frowning face (worst pain). The first face on the scale, which is numbered 0, represents the absence of pain; the next face, numbered 1, represents very little pain; and so forth. The last face on the scale indicates extreme/worst pain. The patient is asked to choose the face that most closely reflects his or her own pain at that point in time (Fig. 12-2). The FPRS is preferred over other pain intensity scales for use with children.[65] The word "hurt," which appears on the FPRS, is the preferred word to use when assessing children, as children may not respond to the word "pain." In an older person, the word "ache" may be more useful in obtaining a response, as some older persons may be stoic and not admit to "pain." The validity and reliability of this scale in adult patients has not been established, although in a geriatric population a high degree of agreement was found between the FPRS and the VAS (r = 92; P < 0.5).[68] The FPRS has been used with cognitively impaired patients and with those for whom English is their second language. A high degree of consistency has been noted between the VAS and FPRS when utilized in the populations tested.

Figure 12-2. FACES Pain Rating Scale. The FACES Pain Rating Scale may be used for children ages 3 and older, for cognitively impaired patients, and for persons less familiar with English. This scale is not always culturally or gender appropriate, as not everyone with a pain score of 10 will cry (Wong-Baker FACES Foundation (2019) Wong-Baker FACES® Pain Rating Scale. Retrieved with permission from http://www.WongBakerFACES.org).

After the initial pain assessment has been completed, reassessment should be performed at regular intervals. Reassess the patient after administration of pain medication or nondrug pain-relieving interventions to ensure that optimal pain relief has been achieved.

Pain Management

Accurate and continuous pain assessment is the foundation of successful pain management.[8,63] However, evidence supports the fact that pain is often poorly assessed. Of physicians with patient care responsibilities in oncology, 76% rated poor pain assessment as the number one barrier to adequate pain management.[11] Donovan et al.[69] found that of the 58% of hospitalized patients reporting excruciating pain, fewer than half had a member of the healthcare team ask them about their pain or note the pain in their records. The use of pain assessment measures has been shown to improve pain management for patients.[68,70] However, problems using the pain assessment scales in everyday practice persist. One problem includes the lack of clinicians' knowledge and familiarity in the use of pain rating scales. Training is required for clinicians to administer pain scales and offer adequate patient instructions on the possible responses to the pain scale questions.

After pain has been identified, its cause should be determined and treated. "The goal of pain management in the pressure injury patient is to eliminate the cause of pain, to provide analgesia, or both".[23] Practical ways of treating pain, depending on the specific chronic wound etiology, have been described by McCaffery and Robinson[4] and Freedman and colleagues.[71]

Dressing changes, débridement, wound edema, infection, turning, and positioning are some of the factors that can cause wound-associated pain. An appropriate plan of action can be implemented after the specific cause of pain has been identified. For example, if the pain results from dressing changes, administering pain medication prior to dressing changes or switching to a different type of dressing may be indicated. According to Bergstrom and colleagues,[24] "Besides medications, pain may be treated with physical and occupational therapy to decrease muscle spasms, decrease contractures, and aide in selecting less painful methods of wound débridement and cleaning. Proper seating, positioning, and adaptive equipment may also help to decrease pain." The optimal way to treat the pain associated with

pressure injuries requires more research, but clinicians can assess the cause of the injury, patient-centered concerns, and all the components of local wound care to minimize pain at each step of their care plan.

Practice Point

Pain management should include interventions that:

- treat the cause
- address patient-centered concerns
- educate the patient
- improve activities of daily living (ADLs) and quality of life
- minimize pain and trauma by incorporating local wound care measures for cleansing, débridement, and moist interactive dressings
- assess and treat critical colonization/deep surrounding infection
- provide palliation to the dying patient
- decrease or eliminate pain with minimal adverse effects
- minimize the patient's dependency on healthcare workers and family members.

Pain Medication

The World Health Organization (WHO)[72] developed a three-step analgesic ladder for the treatment of cancer pain that has been accepted for use in patients with nonmalignant pain[11] (Fig. 12-3). The WHO approach advises clinicians to match the patient's reported pain intensity of 0 to 10 with the potency of the analgesic to be prescribed, starting with nonopioid analgesics for nociceptive pain (acetaminophen, ASA, NSAIDs) and progressing to stronger medications if pain isn't relieved. For example, a patient who reports a pain score of 1 to 3 (mild pain) should receive a nonopioid with or without an adjuvant. If the patient reports a score of 4 to 6 (moderate pain), an appropriate low-dose single or combination opioid with or without an adjuvant should be administered. If the patient's pain score is 7 to 10 (severe pain), he or she should be given a strong opioid with or without an adjuvant. A low-dose single opioid is an opioid given at a low dose that is not combined with another drug within the same pill. An example of a low-dose single opioid is oxycodone 5 mg. A combination opioid is an opioid, which is combined with another drug. An example of a combination opioid is oxycodone with acetaminophen or codeine with acetaminophen. Combination opioids are frequently referred to as "weak opioids," whereas opioids such as morphine, hydromorphone, and fentanyl are referred to as "strong opioids." The clinician should bear in mind that "weak" and "strong" opioids may not apply to all cases, as what is considered weak for one person may be strong for another. For example, an 80-year-old frail person with a pain score of 10/10, who has never used an opioid, may or may not respond to a combination opioid of oxycodone 2.5 mg/acetaminophen 325 mg. Giving a patient an opioid is based not only on the level of pain but also on comorbid conditions, frailty, and previous analgesic history. Any opioid may be potent or cause harm if used indiscriminately or at levels that are inappropriate to the patient and his or her clinical circumstances. An AGS panel reminds us that recommended age-related doses are not available for most analgesics; therefore dosing for most patients requires initiation at low

doses, followed by careful upward titration with frequent reassessment for dosing, pain relief, and side effects.[60,73]

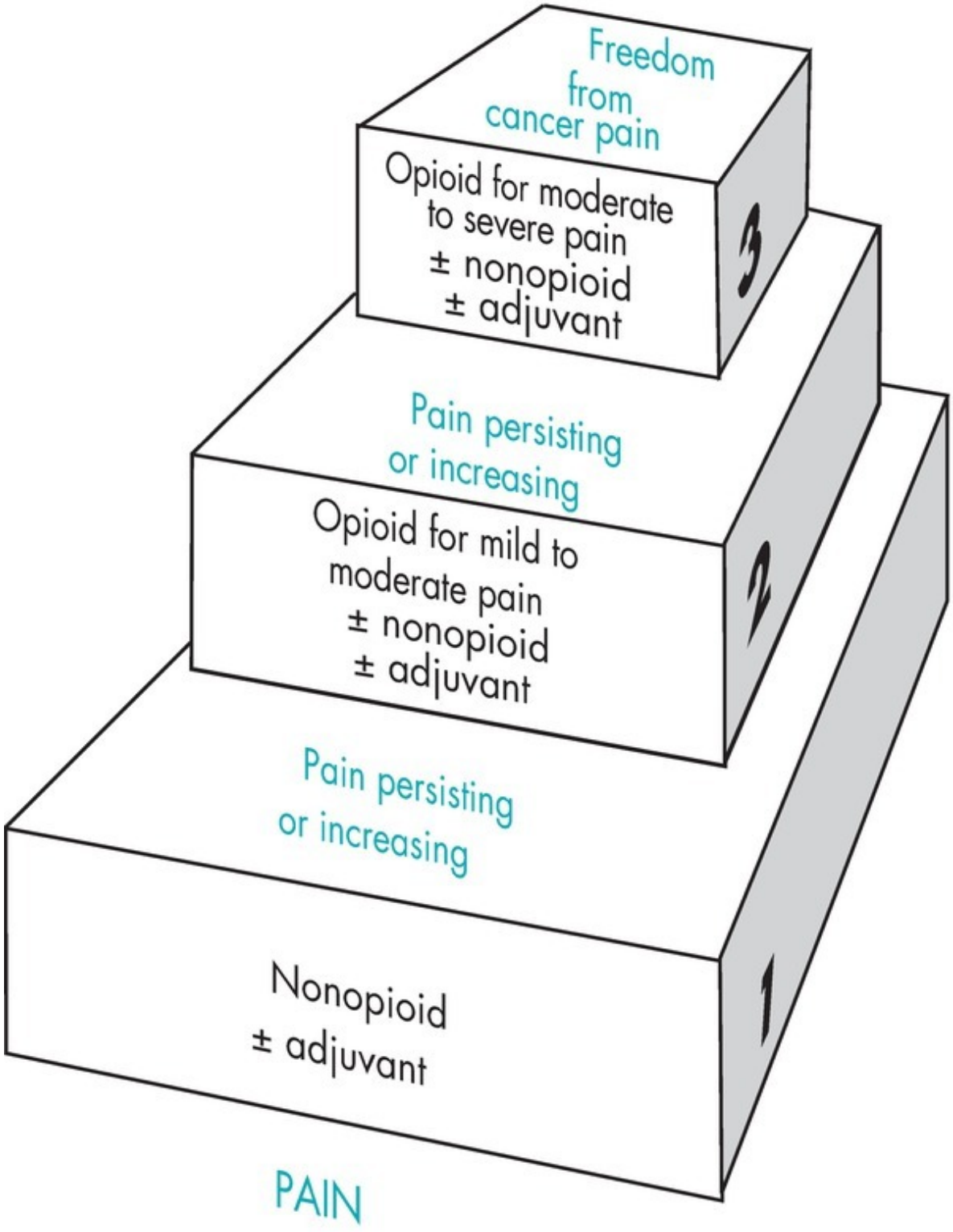

Figure 12-3. WHO analgesic ladder. This analgesic ladder, developed by the World Health Organization (WHO)[72] for pain management in patients with cancer, may be used as a guideline to manage mild through severe wound pain (Copyright © World Health Organization, 2003. Reprinted with permission).

An adjuvant medication is a drug that has a primary indication other than pain but is analgesic for some painful conditions.[8] Examples of adjuvant medications are anticonvulsants or TCA (Table 12-6). Adding an adjuvant medication is most useful in addressing the burning, stinging, shooting, or stabbing symptoms of neuropathic pain. Using a combination of drugs such as an opioid and a nonopioid can enhance pain relief because the two drugs work synergistically. The opioid works on the CNS to alter the perception of pain, and the nonopioid works on the periphery to block painful impulses. Using a combination method may decrease the need for higher doses of opioids.

Table 12-6 Adjuvant Agents

Drug Class	Drug Name	Indications
Tricyclic antidepressants	Amitriptyline Desipramine Nortriptyline	• Multipurpose • Any chronic pain • Lower level of sedation • Neuropathic pain
Anticonvulsants	Carbamazepine Clonazepam Gabapentin Pregabalin Valproic acid	• Burning, neuropathic, lancinating pain
Systemic local anesthetics	Lidocaine Mexiletine	• Gnawing, aching, tender, throbbing
Topical anesthetics	Capsaicin EMLA cream Lidocaine gel Lidocaine 1% Lidocaine 4% Lidocaine patch 5%	• Analgesic for intact skin (use on wound periphery prior to dressing changes) • Before changing vacuum-assisted closure dressing (instill solution through the tubing, with the pressure at 50 mm Hg, and clamp tubing for 15 to 20 minutes)[a] • Saturate gauze for 15 to 20 minutes prior to dressing change • Postherpetic neuralgia • Stump pain

[a]Systemic absorption and toxicity can occur in moderate-to-large wounds. Lidocaine products should not be used in patients who are taking class 1 antiarrhythmic drugs such as tocainide or mexiletine.

Step 1: Nonopioid Analgesics

Acetaminophen or NSAIDs should be initiated as first-line therapy. Acetaminophen is now available as an injectable for IV use. Its indication is for the management of mild-to-moderate pain and with an adjuvant opioid for severe pain. It is useful when parenteral analgesia is clinically warranted, or there is compromise of GI absorption, or an inability to take oral analgesic. It offers 100% bioavailability and can be used in children 2 years of age or older. Parental acetaminophen can be used preoperatively, intraoperatively, or postoperatively. As with PO acetaminophen, it should not be used in doses higher than prescribed given its hepatotoxic profile. Acetaminophen and NSAIDs should be administered on a regular rather than an as-needed basis to increase their effectiveness and maintain a constant level of the medication in the blood. If one group of NSAIDs doesn't work, try another group (Table 12-7).

Table 12-7 Examples of Nonopioid Analgesics

- Acetaminophen
- Aspirin
- Tramadol[a]
- Nonsteroidal anti-inflammatory drugs
  - Celecoxib
  - Ibuprofen
  - Ketorolac
  - Salsalate

[a]Tramadol is not classified as an opioid, but it is centrally acting. It may cause any of the side effects experienced with opioids, including abuse potential and withdrawal. Dose adjustments must be made for those who have renal impairment and/or liver disease. It also lowers seizure thresholds.

NSAID groups include:

- salicylates: aspirin, diflunisal, choline magnesium trisalicylate, salsalate
- propionic acids: naproxen, ibuprofen, fenoprofen, ketoprofen, flurbiprofen, suprofen.
- acetic acids: indomethacin, tolmetin, sulindac, diclofenac
- oxicams: piroxicam.

Clinicians must remember that NSAIDs have increased side effects in older people (especially over 65), including gastrointestinal bleeding, decreased renal function, and aggravation of congestive heart failure, and so should be used with caution in persons over age 65.[73] NSAIDs should also be avoided in patients with platelet disorders such as thrombocytopenia or who are on anticoagulation therapy. NSAIDs are generally not recommended for patients of any age, with kidney disease, heart failure, or cirrhosis or if the patient is using diuretics. There is an increased risk of bleeding in patients with a history of previous esophageal or gastrointestinal bleeding. All NSAIDs,

including gels and patches, carry a black box warning. The warning is as follows:

NSAIDs may cause an increased risk of serious cardiovascular thrombotic events, myocardial infarction, and stroke, which can be fatal. This risk may increase with duration of use. Patients with cardiovascular disease or risk factors for cardiovascular disease may be at greater risk.

Low-dose, single-agent opioids such as oxycodone (5 mg) may be safer for patients with moderate pain who cannot take NSAIDs because of their side effect profile.

TCAs, including amitriptyline, imipramine, nortriptyline, and desipramine, have been shown to relieve neuropathy and postherpetic neuralgia but are contraindicated in patients with coronary disease (although they may be taken cautiously in coronary disease patients at low doses of 10 to 30 mg in a single nighttime dosage).[11] According to an AGS panel, older patients may experience anticholinergic effects of TCAs that may include visual, urinary, gastrointestinal, and cardiovascular changes including orthostatic changes and atrioventricular blockade.[73] Hydroxyzine is a sedating H1 antihistamine that also has analgesic, antiemetic, and mild sedative properties. These drugs may help to induce sleep in patients with chronic pain and are ordered along with other neuropathic agents as adjuvants for nociceptive pain.

Diabetic patients with neuropathic pain or other patients with conditions arising from peripheral nerve syndromes may benefit from the use of certain anticonvulsants, such as gabapentin, pregabalin,[12,13] phenytoin, carbamazepine, sodium valproate, or clonazepam.[11] Clonazepam, although pharmacologically classified as a benzodiazepine, not as an anticonvulsant, has anticonvulsant, muscle relaxant, and anxiolytic properties.

Opioids vary in strength from mild to very strong and are available in different forms, including oral, oral–transmucosal, rectal, transdermal, subcutaneous, IV, and IM.[74,75] The oral form is preferred for long-term use. However, for preprocedural use or for some patients with postprocedural pain from débridement, the IV route may

allow for better pain control and an ability to increase the dosage more quickly as needed. Whether an oral or IV route is used, doses should be scheduled on a regular basis to avoid breakthrough pain. If breakthrough pain occurs when the patient is on a long-acting (sustained-release) opioid regimen, a short-acting (immediate-release) opioid may be given in conjunction with the long-acting opioid to provide pain relief. Breakthrough pain can occur spontaneously but is most likely to occur when the patient is moved, if a dressing change is required, if tubes are being manipulated, or if the patient has an increase in activity. When using opioids around the clock, the clinician should assess the patient for pain caused by an “analgesic gap” or “end of dose failure.” An example of this type of pain is when a patient is on a rescue dose for breakthrough pain every 6 hours as needed, but the medication only provides relief for 4 hours because of its pharmacologic properties. Shortening the interval at which the rescue dose can be used to every 4 hours as needed may correct this problem. Note also that approximately 15% of patients who are on long-acting opioid preparations require dosing every 8 hours rather than every 12 hours (Table 12-8).

Table 12-8 Examples of Combination Opioids

- Acetaminophen with codeine
- Hydrocodone with acetaminophen
- Hydrocodone with ibuprofen
- Oxycodone with acetaminophen
- Oxycodone with ibuprofen

Note that there is a maximum recommended acetaminophen dose to decrease the risk of hepatotoxicity:•Short-term use: (<10 d); 3,000 mg/d•Long-term use: 2,500 mg/d

Constipation can be one of the most common side effects with the use of opioid analgesics. This side effect can be easily remedied by taking stool softeners and laxatives and increasing fiber and fluid

intake (especially water). It's better to anticipate constipation and treat it before it happens.

Other common side effects of opioids include sedation, nausea, vomiting, itching, urinary retention, and sensory or motor deficits. Many clinicians undertreat pain because they fear respiratory depression. While respiratory depression may occur with the use of opioids, it is uncommon in patients who are appropriately dosed and monitored. Patients at risk for developing respiratory depression include those with advanced disease states and/or comorbid conditions such as frailty, chronic obstructive pulmonary disease, congestive heart failure, sleep apnea, and kidney and/or liver disease. Respiratory depression can be decreased or eliminated with the use of lower initial doses that are increased gradually while monitoring the patient's vital signs, especially the quality and rate of respiration and the patient's sedation status.

Evidence-Based Practice

Avoiding Addiction: The Four A's of Opioid Treatment

Caregivers tend to underutilize opioid analgesics because of the fear of **addiction.** Caregiver education in this area is very important, especially in the home care setting where the caregiver is the one deciding when to give the patient pain medication. Addiction to opioids should be a concern, but the fear of addiction has been greatly exaggerated. Studies have shown that the incidence of addiction in patients who take an opioid for acute pain relief is about 1%. The length of time on the analgesic and the amount given are irrelevant to the risk for addiction.[26]

While the incidence of addiction is low, the potential for **abuse** does exist. Therefore, clinicians should remain diligent in monitoring patients who are taking opioids to assess their risk for abuse.

Patients with a history of nonopioid substance abuse, including cigarettes and alcohol, and mental health disorders are at higher risk of developing opioid abuse.[75] In addition, there are reports of rising prescription drug abuse with greater use of opioids for the treatment of chronic pain.[76–78] These types of reports have prompted the FDA to develop a multiagency federal effort to address Risk Evaluation and Mitigation Strategies (REMS) for extended-release and long-acting opioids.[79] These strategies include proposed class wide labeling changes that will help to insure their safe and appropriate use.[80] In keeping with the paradigm of protecting patients from potential harm associated with chronic opioid use, the AGS panel and others recommend that patients be assessed for risk factors related to problematic use of pain medications by using initial assessment tools such as the ORT (Opioid Risk Assessment Tool) or the SOAPP-R (Screener and Opioid Assessment for Patients with Pain-Revised).[73,76] An indication that abuse is developing is if the patient begins to crave the drug for reasons other than pain relief. When patients are using opioids, it is important for the clinician to assess and document the 4 A's of opioid treatment as follows:

1. **Analgesia** (Is the pain score going down?)
2. **Activities of daily living** (Is there improvement in functional ability, positioning?)
3. **Adverse effects** (Are there mental status changes, such as confusion, or other effects, such as sedation, jerky motions, etc.?)
4. **Aberrant behaviors** (Does the patient report lost prescriptions, lost pills?)

Meperidine is not recommended for the management of persistent (chronic) pain. Disadvantages include the hazards of the normeperidine metabolite of meperidine. Repeated doses of meperidine can lead to an accumulation of normeperidine, which causes CNS excitability and toxicity. This toxicity will be manifested in the patient by twitching, numbness, seizures, and hallucinations.[2] Coma and death are also possible. According to the APS,[11] "although

the oral doses of meperidine have about one quarter of the analgesic effectiveness of similar parenteral doses, they produce just as much of this toxic metabolite." The APS also warns that patients with compromised renal function are particularly at risk for the accumulation of this toxic metabolite.

Step 2: Opioids for Mild-to-Moderate Pain

Opioids combined with an NSAID, or acetaminophen, or low-dose single-agent opioids, such as oxycodone immediate-release 5 mg, may be used if step 1 is ineffective (Table 12-8). Propoxyphene has been withdrawn because of its cardiac toxicity profile. The FDA has concluded that the safety risk of propoxyphene outweighs its benefits for pain relief at the recommended doses and advised healthcare professionals to stop prescribing and dispensing it. The generic Darvon is no longer available in the United States. Codeine may cause excessive constipation, nausea, and vomiting.[63]

Step 2: Opioids for Moderate-to-Severe Pain

Low-dose opioids, including morphine and morphine-like agents, may be added to step 1 when it is ineffective (Table 12-9). An adjuvant drug such as the tricyclic agents (nortriptyline, desipramine) or antiepileptics (gabapentin, pregabalin) may also be used, if there is concurrent neuropathic pain, as well as somatic or visceral pain. The prescriber as well as the nurse caring for the patient should be aware of potential side effects and possible drug interactions. This includes knowledge of age and disease-related potential hazards and risks of all drugs used.

Table 12-9 Examples of Opioids: Morphine and Morphine-Like Agents

- Morphine
- Methadone
- Fentanyl
- Hydromorphone
- Levorphanol
- Oxycodone

Practice Point

Helpful Hints for Using Opioids

- Fentanyl
  - Oral–transmucosal fentanyl works in 10 minutes, which is convenient for dressing changes, but its use for this purpose is considered off-label. This drug is FDA approved only for cancer-related pain. Oral–transmucosal fentanyl should only be used in patients who are opioid tolerant (patients whose total dose of opioids is equal to 60 mg of morphine a day, for a total of 1 week or longer).
  - Fentanyl patches should be started at the lowest dose for patients who are opioid naive (have never used opioids). Opioid-naive patients need careful monitoring. In frail patients, even the lowest dose may cause sedation or other unwanted side effects.
  - It will take 18 to 24 hours for the first patch to reach maximum effect. If immediate pain relief is required, a short-acting opioid can be used along with the patch and then discontinued, if needed,

when the patch begins to reach its maximum effect. A rescue dose of a short-acting opioid may still be needed for breakthrough pain once the long-acting dose has been titrated.

- Use short-acting agents (at 10% to 15% of total daily dose) for breakthrough pain.
- If a patient is on both a long-acting agent and a short-acting (immediate-release) agent for breakthrough and both agents are opioids for severe pain, the long-acting dose may needed to be titrated up, if the patient remains in pain over time, is without side effects, and is requiring more than three breakthrough doses daily.
- Don't wait for constipation to begin. Start the patient on stool softeners and laxatives to avoid this common adverse effect.
- To taper or discontinue opioid analgesics, decrease the dose by 25% every 2 to 3 days. Monitor for pain or withdrawal symptoms, as these would indicate that the tapering is too rapid.

Morphine is one of the drugs of choice for chronic pain largely because it is more cost-effective than meperidine, may be administered via many different routes, and is easily titrated. It has predictable and treatable side effects. The side effects of nausea and constipation are usually treated with antiemetic drugs and laxatives, respectively.

A pegylated derivative of the mu-opioid receptor agonist naloxone (naloxegol) has been used to treat opioid-induced constipation with promising results. Other side effects are usually dose related and can be resolved with dose adjustments.[8] Some patients cannot tolerate the side effects of morphine even with dose adjustments, and some experience significant side effects such as a skin rash or changes in mental status; the drug should be discontinued in these patients, and a pain management consultation should be sought to determine an appropriate alternative analgesic.

Morphine is not for use in patients with renal disease (glomerular filtration rate <30 mL/min) owing to the rapid accumulation of nondialyzable metabolite that can be neurotoxic.[81] Patients who have liver disease may also be sensitive to the effects of morphine because it takes longer for the damaged liver to metabolize and eliminate the drug. Patients with asthma may have increased wheezing with morphine use and should be monitored carefully. Any patient whose daily dose of narcotic agents exceeds the equivalent of 120 mg of morphine should have a specialized pain consultation.[74]

Although studies are limited, some researchers are exploring the effects of topical opioids in the treatment of painful skin ulcers. In their report on nine patients with open skin ulcers caused by a variety of medical conditions, Twillman and colleagues[82] documented that pain at the ulcer site was decreased when a morphine-infused gel dressing was used. The researchers reported remarkable efficacy in eight of the nine patients studied and believe further research is needed in this area because so many patients stand to gain pain relief.

Nonpharmacologic Treatment Modalities

Management of pain from wounds can require a combination of pharmacologic and nonpharmacologic treatments; the latter may include the use of music, massage, and relaxation techniques. Pain associated with dressing changes and débridement can be minimized by allowing patients to call “time-outs.” Many other nonpharmacologic treatments can also be used prior to, and in conjunction with, medications. These include physical and occupational therapy, repositioning the patient, providing support surfaces, and optimizing local wound care with materials that minimize pain.

Practice Point

Symptoms of withdrawal or abstinence from opioids include:

- tachycardia
- hypertension
- insomnia
- diaphoresis
- piloerection
- enlarged pupils
- nausea and diarrhea
- abdominal pain
- body aches
- increased sensitivity to pain
- yawning
- runny nose
- anxiety.

The presence of these symptoms may indicate that the opioid medication is being tapered too quickly and not necessarily that the

patient is addicted to it.

Physical and Occupational Therapy

Physical and occupational therapy services may be a valuable asset to utilize in conjunction with pharmacologic therapy. Passive and active range-of-motion exercises should be taught to the patient and his or her caregivers. Additional measures include the following:

- Patients with peripheral vascular disease may benefit from a walking program to facilitate development of collateral circulation in the lower extremities.
- Application of a transcutaneous electrical nerve stimulation unit may help to decrease pain, particularly in patients with chronic or acute wounds. It's believed that the electrical stimulation provided by the unit helps to inhibit pain transmission cells.
- Hot or cold packs can be applied to decrease spasms in the affected area.
- Stretching exercises help to decrease contractures.
- Exercise helps to decrease muscle spasms with massage.

Local Pain Management

The use of appropriate dressings and dressing techniques can help to relieve pain during and between dressing changes. The international survey conducted by Moffatt et al.[45] highlights the importance of low-pain dressing changes. All respondents agreed that gauze dressings cause the most pain, while pain is noticeably less severe with the use of hydrogels, hydrofibers, alginates, and soft silicone dressings.

When administering wound care, choose products carefully to provide the patient with a pain-free experience. (See Chapter 9, Wound Treatment Options.) Patients who express discomfort despite careful product selection should be given medication prior to dressing changes.

Moist wound dressings can be left in place for a longer period of time than wet-to-dry dressings. This reduces the frequency of

dressing changes, thereby decreasing the opportunities for the patient to experience pain associated with dressing changes.

Practice Point

Wet-to-dry dressings can desiccate a wound, thus causing pain on removal. (Removing viable stuck tissue from the wound surface leads to bleeding, trauma, pain, and delayed wound healing.) Avoid these dressings in favor of moisture-retentive dressings to promote a healing environment and patient comfort.

The following interventions will ease the dressing change process and manage pain for your patients.

Treating the Cause/Aggravating Factors

- Provide pressure redistribution for your patients.
- Keep the patient's heels off the bed at all times.
- Control edema to avoid decreased blood flow to the wound, which may lead to additional pain.
- Eliminate or decrease pain from other possible pain sources.

Addressing Patient-Centered Concerns

- Keep in mind that pharmacologic management is the gold standard for moderate-to-severe pain. Give pain medication around the clock, if necessary, to keep the pain under control.
- Instruct the patient and his or her family regarding pain management to alleviate fear of addiction with the use of opioids.
- Explain the role that pain control plays in improved wound healing.

- Outline the dressing change procedures to the patient before proceeding with dressing changes.
- Allow the patient to select the time for dressing changes, if appropriate.
- Assess for pain and medicate the patient before and after dressing changes or débridement.
- Invite the patient or family members to participate in dressing changes, as indicated.
- Offer the patient distraction techniques, such as conversation, television, and videos during dressing changes.
- Inform the patient that he or she may call a “time-out” if pain is present during dressing changes.
- Ensure that the patient has adequate rest and sleep. Lack of rest and sleep will decrease the patient’s pain threshold, decrease his or her mental performance, and increase the emotional response to pain.
- Teach the patient to substitute worry beads, a pet rock, bean bag, tapping, rubbing, or gentle slapping for scratching.
- Instruct the patient in relaxation techniques and the use of visual imagery when encountering a potentially pain-provoking situation.
- Reevaluate the pain management plan when needed. Document the effectiveness of the analgesic or other treatments for pain relief with a pain score. This will help to assess whether the pain management program is working.
- Address other factors, such as loss of function, inability to perform ADLs, and possible changes in body image to help the patient deal with ancillary problems that might contribute to his or her pain.

Cleansing and Débridement

- Assess pain when the patient is at rest to provide adequate pain control.
- Apply warm normal saline solution or low cytotoxicity wound cleaners to clean wounds. (Cytotoxic solutions such as (Betadine) iodine solutions or hydrogen peroxide not only

deter wound healing, but they may cause burning, adding to the patient's discomfort.)

- Use moist wound therapy to enhance autolytic débridement as an alternative to surgical or sharp débridement to eliminate pain associated with sharp débridement.
- Change dressings in a timely manner. Excess exudate on periwound skin or dressings that are allowed to dry on a wound may increase the patient's pain.
- Protect the periwound skin with skin barrier wipes, film-forming liquid acrylates, or ointment (petrolatum or zinc oxide) to prevent excoriation, trauma, maceration, or dermatitis that can delay wound healing, increase wound size, and increase patient discomfort. Avoid using strong adhesive tape on elderly patients or patients with fragile skin.

Alternative Pain Management Methods

Many natural pain control methods and therapies may be implemented to ease pain, stress, and anxiety. These methods can improve one's outlook, attitude, and quality of life. Alternative therapies, when used in conjunction with pain medications, may enhance the beneficial effects of pain medication.

Laughter. Laughter helps you breathe deeper, lowers your blood pressure, and changes your mood.

Acupuncture. The application of needles to specific areas of the body may decrease or eliminate pain and has been used for more than 2,500 years.

Environment. Having the room at a comfortable temperature, avoiding bright lights, and keeping the room quiet may help to decrease pain.

Distraction. Playing cards, watching television, visiting with friends, petting an animal, and writing about his or her feelings can help the patient focus attention on something other than the pain.

Music. Music increases blood flow to the brain and increases energy, which in turn causes an increase in the production of endorphins (a natural body chemical similar to morphine) that work to decrease or eliminate pain and anxiety.

Magnets. Magnets may effect changes in cells or body chemistry that can produce pain relief. The use of magnets, which dates back to ancient Egypt and Greece, is popular with athletes, who report their effectiveness in controlling pain.

Capsaicin. Capsaicin, a chemical found in chili peppers, is the primary ingredient in many pain-relieving creams for the treatment of neuropathic pain. The local burning sensation it produces replaces the pain sensation.

Summary

Pain scores derived from patient-completed validated pain assessment scales can be useful as the basis for assessing and treating chronic wound-associated pain. The scales enable the clinician to accurately assess the patient's pain, thereby facilitating effective treatment modalities to help decrease the wound-associated pain. Pain is detrimental for patients because it can exhaust them, reduce their ability to perform ADLs, add to feelings of decreased worth as a person, affect their interactions with loved ones and friends, deter wound healing, and, overall, diminish quality of life as well as quality of death for patients who are in the process of dying. As clinicians, we are obligated to provide adequate pain relief for our patients by selecting appropriate pain control treatment modalities.

PATIENT SCENARIO

Clinical Data

Mr. LK is a 67-year-old male who is hospitalized for infected leg ulcer. He reports pain associated with this ulcer. His self-report pain level is 10/10. The pain extends beyond the wound edges and is present during rest and increases with dressing changes. He describes the pain as deeply aching down to the muscle with unbearable burning as if he was burning alive. He states he is "saddened and terrified by the burning feeling" and further describes it as the way that his "buddies must have felt when they were wounded or killed during the war." He further adds "I didn't burn then, but I am burning now."

The leg ulcer has been present for 9 months, progressively worsening. The leg wound is a shallow 4 × 6 cm on the lateral aspect of the lower leg. The wound bed is composed of red, friable granulation tissue and yellowish, malodorous slough on the surface. The wound is associated with edema, which worsens at the end of the day; inflammatory woody fibrosis; and surrounding cellulitis. The

patient reports increased exudate and odor. The pain affects his walking, sleeping, and performing ADLs.

Mr. LK lives alone. He is a Vietnam War veteran. He is a one-half pack day smoker for 40 years and continues to smoke “3 to 4 cigarettes a day.” He admits to a remote history of marijuana use and denies illicit and/or prescription drug use/abuse. He reports he is fearful of opioids because of their additive properties. He drinks one to two beers on the weekends but abstains from hard liquor. He has had no psychiatric comorbid conditions but states he sometimes suffers “pain of his soul.” He does not use sleeping medications, benzodiazepines, or any other sedating medications or psych meds. Mr. LK’s other comorbid conditions include hypertension, hyperlipidemia, osteoarthritis, stage III chronic kidney disease, and a history of DVTs. His analgesic history is as follows:

- Acetaminophen with 30% relief
- Codeine with acetaminophen with 30% relief
- Tramadol with adverse effects of confusion and having to stop medications
- Short-acting morphine 15 mg every 8 hours PRN with 60% relief, which lasts only 4 hours past dosing

Mr. LK has a nonhealing painful ulcer that is infected and associated with surrounding cellulitis, consistent with a venous ulcer.

Case Discussion

Initial Pain Assessment and Treatment Rationale

Mr. LK should avoid the use of codeine and morphine as its metabolite can accumulate in patients with renal disease and cause neurotoxicity and respiratory depression. Likewise, NSAIDs should be used with caution because they can aggravate renal disease.

The recommended treatment for this patient is to use a combination of pain relief modalities, such as drug- and non–drug-related modalities. Because this patient is fearful of opioid use, it is important to educate the patient about the risks, benefits, and alternatives to opioid use. Every effort should be made in using multimodal strategies and minimizing the use of opioids. If an opioid must be used, it should be used at the lowest dose possible, which is consistent with pain relief and maximizing function.

Nondrug Strategies

The wound is treated locally for reducing the bioburden and controlling the exudate with cleansing of debris and autolytic débridement. The surrounding cellulitis is treated with systemic antibiotics. Reducing infection and exudate is helpful in pain reduction. The patient is instructed to avoid long periods of sitting to avoid pooling of fluids in the leg; when sitting the patient should elevate the leg, as edema increases the pain locally. Smoking cessation was also discussed with the patient. Wet-to-dry dressing and strong adhesive tapes are avoided, because these can increase pain at dressing change. A Doppler study was performed, given the report of muscle pain and history of DVTs, and found to be negative.

As there was some concern related to possible psychosocial issues and unresolved emotional components of pain (pain of the soul) and possibly survival guilt (I didn’t burn then, but am burning now) and tearfulness and reports of sadness, the patient agreed to a psych evaluation.

Nonopioid Strategies

To ameliorate the burning sensation in and around the ulcer, treatment should include a TCA, starting at the lowest dose possible, or gabapentin starting at 100 mg at bedtime and slowly titrated to 300 mg daily (renal dosed based on creatinine clearance).

The patient asked if he could take acetaminophen for his arthritis pain instead of using the oxycodone and was instructed that he should avoid drinking alcohol if he wanted to use acetaminophen and should not exceed 1,000 mg every 8 hours a day (3,000 mg/d) because of the hepatotoxic effects. This includes taking into account all other products such as allergy or cold medications that contain acetaminophen that he may also be using. The patient is reminded that the FDA acetaminophen manufacturers have warned that having three drinks a day while using acetaminophen can cause severe liver damage.

Opioid Strategies

For his chronic pain, a low-dose fentanyl patch 12 µg was ordered because the patient reported a pain score of 10/10 affecting his ability to sleep, ambulate, and perform ADLs. A pain score of 10/10 is considered severe pain and requires rational use of opioids for pain relief when other treatment strategies have failed. The fentanyl patch is long acting and provides around-the-clock relief and is changed every 72 hours. Prior to starting the opioid treatment, a urine toxicology screen was performed and found to be negative. The patient was given an Opioid Risk Tool (ORT) and scored 2/10. The patient signed an opioid use agreement, covering his knowledge of risks, benefits, and alternate treatments and safety issues related to safely using and storing the opioid patches.

Because this patient's pain is mostly chronic, it requires communicating realistic expectations. It is important to discuss that complete freedom from pain may not be achievable. The focus should be on increasing function and mobility and relieving associated psychological stressors as well as minimizing the risk of opioid misuse, abuse, and addiction that can be associated with long-term analgesic opioids.

For breakthrough pain, oxycodone 5 mg (without acetaminophen) was ordered every 6 hours PRN.

Follow-Up Pain Assessment and Treatment

The patient was assessed for pain and functional status. He was examined and found to be using the fentanyl patch that was located on his right upper chest and requiring the oxycodone only prior to dressing change. He reported a pain score of 3/10 with the use of the fentanyl patch and the gabapentin titrated up to 300 mg twice a day. He reported that he could live with this pain level.

After the infection cleared, the patient did not require the acetaminophen but said he would still use it for his arthritis pain and would like to try it prior to dressing change to see if it would be helpful so that he could avoid the oxycodone altogether. He reported that he was sleeping and walking better and able to get up and down the stairs with less discomfort. The oxycodone was titrated down to 5 mg, 30 minutes prior to dressing change. The patient was started on a compression dressing.

The patient was evaluated by psychiatry and was not found to have a major depressive disorder but agreed to attend the chronic pain support group available at the hospital and reported that it was helpful to feel that he was not alone with his pain. Opioid-induced constipation was treated with a combination of Colace, Senna, and Dulcolax.

Show What You Know

1. **Which of the statements listed below most accurately defines pain? Pain is:**
   A. an objective finding based on prolonged elevation of the patient's blood pressure and pulse rate.
   B. a state of discomfort evidenced by the person being unable to sleep.
   C. a physical consequence of wound care.
   D. whatever the experiencing person says it is.

2. **Which of the following statements best describes the Numeric Pain Intensity Scale? It is a:**
   A. 10-cm line with the words "no pain" at one end and "worst possible pain" at the other end.
   B. series of faces ranging from smiling to frowning.
   C. rainbow of colors starting with green and ending with red.
   D. decision tree for determining which medications to give to a person experiencing pain.

3. **According to the WHO analgesic ladder, which medications should you use initially for relief of mild pain?**
   A. None
   B. Nonopioid with or without an adjuvant
   C. Opioid with or without an adjuvant
   D. Opioid

***Answers to these questions are found on page* 684.**

References

1. “Pain Terms: A List with Definitions and Notes on Usage Recommended by the IASP Subcommittee on Taxonomy.” Pain 6(3):249-52, June 1979.
2. Acute Pain Management Guideline Panel. Acute Pain Management: Operative or Medical Procedures and Trauma, Clinical Practice Guideline, No. 3. Rockville, MD: AHCPR, 1992.
3. McCaffery, M., Beebe, A. Pain: Clinical Manual for Nursing Practice. St. Louis, MO: Mosby–Year Book, Inc., 1989.
4. McCaffery, M., Robinson, E.S. “Your Patient Is in Pain, Here’s How You Respond.” Nursing 32(10): 36-45, October 2002.
5. Dahl, J.L., Gordon, D.B. “Joint Commission Pain Standards: A Progress Report,”. American Pain Society Bulletin 12(6), 2002.
6. American Pain Society. “Pain: The Fifth Vital Sign,”. 1995. Retrieved May 5, 2014 from http://www.ampainsoc.org/advocacy/fifth.htm
7. Frescos, N. “Assessment of Pain in Chronic Wounds: A Survey of Australian Health Care Practitioners.” International Wound Journal 15(6):943-9, 2018. Available at: https://doi.org/10.1111/iwj.12951
8. McCaffery, M., Passero, C. Pain: Clinical Manual, 2nd ed. St. Louis, MO: Mosby–Year Book, Inc., 1999.
9. Taverner, T., Closs, S.J., Briggs, M. “The Journey to Chronic Pain: A Grounded Theory of Older Adults’ Experiences of Pain Associated with Leg Ulceration.” Pain Management Nursing 15(1):186-98, 2014. Available at: https://doi.org/10.1016/j.pmn.2012.08.002.
10. Moulin, D., Boulanger, A., Clark, A.J., et al. “Pharmacological Management of Chronic Neuropathic Pain: Revised Consensus Statement from the Canadian Pain Society.” Pain Research and Management 19(6):328-35, 2014. Available at: https://doi.org/10.1155/2014/754693.
11. American Pain Society. Principles of Analgesic Use in the Treatment of Acute Pain and Cancer Pain, 6th ed. Glenview, IL: Author, 2008.
12. Lesser, H., et al. “Pregabalin Relieves Symptoms of Painful Diabetic Neuropathy: A Randomized Controlled Trial.” Neurology 63(11):2104-10, December 2004.
13. Dworkin, R.H., et al. “Pregabalin for the Treatment of Postherpetic Neuralgia: A Randomized, Placebo-controlled Trial.” Neurology 60(8):1274-83, April 2003.
14. AGS Panel on Persistent Pain in Older Persons. “The Management of Persistent Pain in Older Persons.” Journal of American Geriatric Society 50(6 Suppl):S205-24, June 2002.
15. Arroyo-Novoa, C.M., Figueroa-Ramos, M.I., Miaskowski, C., et al. “Acute Wound Pain: Gaining a Better Understanding.” Advances in Skin & Wound Care 322(8):373-80, 2009.

16. Polomano, R.C. “Neurophysiology of Pain.” In B. St. Marie, ed. Core Curriculum for Pain Management Nursing, 2nd ed. Philadelphia, PA: WB Saunders Company, 2002.
17. Dallam, L., et al. “Pressure Ulcer Pain: Assessment and Quantification.” Journal of Wound, Ostomy, and Continence Nursing 22(5):211-7, September 1995.
18. Krasner, D. “The Chronic Wound Pain Experience: A Conceptual Model.” Ostomy/Wound Management 41(3):20-9, April 1995.
19. Krasner, D. “Caring for the Person Experiencing Chronic Wound Pain.” In D.L. Krasner, ed. Chronic Wound Care: A Clinical Source Book for Healthcare Professionals, 3rd ed. Wayne, PA: HMP Communications, 2001.
20. Krasner, D. “Managing Wound Pain in Patients with Vacuum-Assisted Closure Devices.” Ostomy/Wound Management 48(5):38-43, May 2002.
21. Gardner, S.E., Frantz, R.A., Doebbeling, B.N. “The Validity of the Clinical Signs and Symptoms Used to Identify Localized Chronic Wound Infection.” Wound Repair and Regeneration 9:178-86, 2001.
22. National Pressure Ulcer Advisory Panel. “Pressure Ulcer Prevalence, Cost, and Risk Assessment: Consensus Development Conference Statement.” Decubitus 2(2):24-8, May 1989.
23. van Rijswijk, L., Braden, B.J. “Pressure Ulcer Patient and Wound Assessment: An AHCPR Clinical Practice Guideline Update.” Ostomy/Wound Management 45(1A Suppl):56S-67, January 1999.
24. Bergstrom, N., et al. Pressure Ulcer Treatment: Clinical Practice Guideline #15. Rockville, MD: AHCPR, 1994.
25. Pieper, B. “Mechanical Forces: Pressure, Shear and Friction.” In R.A. Bryant, ed. Acute and Chronic Wounds: Nursing Management, 2nd ed. St. Louis, MO: Mosby–Year Book, Inc., 2000.
26. Rook, J.L. “Wound Care Pain Management.” Advances in Wound Care 9(6):24-31, November-December 1996.
27. Szors, J.K., Bourguignon, C. “Description of Pressure Ulcer Pain at Rest and Dressing Change.” Journal of Wound, Ostomy, and Continence Nursing 26(3): 115-20, May 1999.
28. Pieper, B., Langemo, D., Cuddigan, J. Pressure Ulcer Pain: A Systemic Literature Review and National Pressure Ulcer Advisory Panel White Paper. Available at: http://www.npuap.org/wp-content/uploads/2012/01/Pieper_2009_Feb1.pdf. Last accessed 27 July, 2014.
29. Langemo, D., Black, J., the NPUAP. “Pressure Ulcers in Individuals Receiving Palliative Care: A NPUAP White Paper.” Advances in Skin & Wound Care 23(2):59-72, February 2010.
30. Gerhard-Herman, M.D., Gornik, H.L., Barrett, C., et al. AHA/ACC Guideline on the Management of Patients With Lower Extremity Peripheral Artery Disease: A Report of the American College of Cardiology/American Heart

Association Task Force on Clinical Practice Guidelines. Journal of the American College of Cardiology. 2016 Nov 8. pii: S0735-1097(16)36902-9.
31. Newbern, S. “Identifying Pain and Effects on Quality of Life from Chronic Wounds Secondary to Lower-Extremity Vascular Disease: An Integrative Review.” Advances in Skin & Wound Care 31(3):102-8, 2018.
32. Sibbald, R.G., Woo, K., Ayello, E.A. “Increased Bacterial Burden and Infection: The Story of NERDS and STONES.” Advances in Skin & Wound Care 19(8):447-62, 2006.
33. Sibbald, R.G., Ovington, L.G., Ayello, E.A., et al. “Wound Bed Preparation 2014 Update: Management of Critical Colonization with a Gentian Violet and Methylene Blue Absorbent Antibacterial Dressing and Elevated Levels of Matrix Metalloproteases with an Ovine Collagen Extracellular Matrix Dressing.” Advances in Skin & Wound Care 27(1 Suppl):1-6, March 2014.
34. Marin, J.A., Woo, K.Y. Clinical Characteristics of Mixed Arteriovenous Leg Ulcers: A Descriptive Study. Journal of Wound, Ostomy, and Continence Nursing 44(1):41-7, Jan/Feb 2017.
35. deJong, A.E., Bremer, M., van Komen, R., et al. Pain in Young Children with Burns: Extent, Course & Influencing Factors. Burns 40:38-47, 2014.
36. Pardesi, O., Fuzaylov, G. “Pain Management in Pediatric Burn Patients: Review of Recent Literature and Future Directions.” Journal of Burn Care & Research 38(6):335-47, 2017. Available at: https://doi.org/10.1097/BCR.0000000000000470.
37. Sarthak, S., Schreiner, A.J., Biernaskie, J., et al. “Treating Pain on Skin Graft Donor Sites: Review and Clinical Recommendations.” Journal of Trauma and Acute Care Surgery 83(5):954-64, 2017. Available at: https://doi.org/10.1097/TA.0000000000001615.
38. Alavi, A., Anooshirvani, N., Kim, W.B., et al. Quality-of-life Impairment in Patients with Hidradenitis Suppurativa: A Canadian Study. American Journal of Clinical Dermatology 16(1):61-5, Feb 2015. doi: 10.1007/s40257-014-0105-5
39. Horváth, B., Janse, I.C., Sibbald, G.R. “Pain Management in Patients with Hidradenitis Suppurativa.” Journal of the American Academy of Dermatology 73(5):S47-51, 2015. Available at: https://doi.org/10.1016/j.jaad.2015.07.046.
40. Hollingworth, H. “Nurse’s Assessment and Management of Pain at Wound Dressing Changes.” Journal of Wound Care 4(2):77-83, February 1995.
41. European Wound Management Association. EWMA Position Document: Pain at Wound Dressing Changes. London, UK: MEP Ltd., 2002.
42. Briggs, M., Ferris, F.D., et al. Principles of Best Practice: A World Union of Wound Healing Societies’ Initiative. Minimizing Pain at Wound Dressing-Related Procedures: A Consensus Document. London, UK: MEP Ltd., 2004.
43. Latarjet, J. “The Management of Pain Associated with Dressing Changes in Patients with Burns.” World Wide Wounds (Electronic Journal), November 2002.

44. Krasner, D. “Using a Gentler Hand: Reflections on Patients with Pressure Ulcers Who Experience Pain.” Ostomy/Wound Management 42(3):20-9, April 1996.
45. Moffatt, C.J., Franks, P.J., Hollinworth, H. “Understanding Wound Pain and Trauma: An International Perspective.” In S. Calne, ed. EWMA Position Document: Pain at Wound Dressing Changes. London, UK: MEP Ltd., 2002:2-7.
46. Wulf, H., Baron, R. “The Theory of Pain.” In S. Calne, ed. EWMA Position Document: Pain at Wound Dressing Changes. London, UK: MEP Ltd., 2002:8-11.
47. Briggs, M., Torra I Bou, J.E. “Pain at Wound Dressing Changes: A Guide to Management.” In S. Calne, ed. EWMA Position Document: Pain at Wound Dressing Changes. London, UK: MEP Ltd., 2002:12-7.
48. Woo, K. Wound-Related Pain and Attachment in the Older Adults. Saarbrucken, Germany: Lambert Academic Publishing, 2002.
49. Upton, D., Solowiej, K. “Pain and Stress as Contributors to Delayed Wound Healing.” Wound Practice and Research 18(3):114-22, 2010.
50. Solowiej, K., Mason, V., Upton, D. “Psychological Stress and Pain in Wound Care, Part 2: A Review of Pain and Stress Assessment Tools.” Journal of Wound Care 19(3):110-5, 2010. Available at: https://doi.org/10.12968/jowc.2010.19.3.47280.
51. Franks, P.J., Collier, M.E. “Quality of Life: The Cost to the Individual.” In M.J. Morrison, ed. The Prevention of Pressure Ulcers. St. Louis, MO: Mosby–Year Book, Inc., 2001.
52. Ayello, E.A., et al. Is Pressure Ulcer Pain Documented? Paper presented at the Clinical Symposium on Advances in Skin and Wound Care, Las Vegas, NV, 2006.
53. Langemo, D.K., et al. “The Lived Experience of Having a Pressure Ulcer: A Qualitative Analysis.” Advances in Skin & Wound Care 13(5):225-35, September-October 2000.
54. Rastinehad, D. “Pressure Ulcer Pain.” Journal of Wound, Ostomy, and Continence Nursing 33(3):252-7, May-June 2006.
55. Hopkins, A., et al. “Patient Stories of Living with a Pressure Ulcer.” Journal of Advanced Nursing 56(4): 1-9, April 2006.
56. Roth, R.S., Lowery, J.C., Hamill, J.B. “Assessing Persistent Pain and Its Relation to Affective Distress, Depressive Symptoms, and Pain Catastrophizing in Patients with Chronic Wounds: A Pilot Study.” American Journal of Physical Medicine & Rehabilitation 83(11):827-34, November 2004.
57. Shukla, D., Tripathi, A.K., et al. “Pain in Acute and Chronic Wounds: A Descriptive Study.” Ostomy/Wound Management 51(11):47-51, November 2005.

58. Fox, C. “Living with a Pressure Ulcer: A Descriptive Study of Patients’ Experiences.” British Journal of Community Nursing 7:10-22, July 2002.
59. Ayello, E.A. “Teaching the Assessment of Patients with Pressure Ulcers.” Decubitus 5(4):53-4, July 1992.
60. Ayello, E.A. “A Pressure Ulcer ASSESSMENT Tool.” Advances in Skin & Wound Care 13(5):247, September-October 2000.
61. Herr, K., et al. “Pain Assessment in the Nonverbal Patient: Position Statement with Clinical Practice Recommendations.” Pain Management Nursing 17(2): 44-52, June 2006.
62. Hartford Institute for Geriatric Nursing. Try This. Retrieved May 23, 2014 from http://www.hartfordign.org
63. Fink, R., Gates, R. “Pain Assessment.” In B.R. Ferrell, N. Coyle, ed. Textbook of Palliative Nursing. New York: Oxford University Press, 2001.
64. Keele, K.D. “The Pain Chart.” The Lancet 48(2):6-8, February 1948.
65. Wong, D.L., Baker, C.M. “Pain in Children: Comparison of Assessment Scales.” Pediatric Nursing 14(1):9-17, January-February 1988.
66. Flaherty, S.A. “Pain Measurement Tools for Clinical Practice and Research.” AANA Journal 64(2):133-40, April 1996.
67. Hockenberry-Eaton, M., Wilson, D. Wong’s Essentials of Pediatric Nursing, 8th ed. St. Louis, MO: Mosby, 2009.
68. Simon, W., Malabar, R. “Assessing Pain in Elderly Patients Who Can’t Respond Verbally.” Journal of Advanced Nursing 22(4):663-9, October 1995.
69. Donovan, M., et al. “Incidence and Characteristics of Pain in a Sample of Medical-Surgical Patients.” Pain 30(1):69-78, July 1987.
70. Faires, J.E., et al. “Systematic Pain Records and Their Impact on Pain Control: A Pilot Study.” Cancer Nursing 12(6):306-13, December 1991.
71. Freedman, G., et al. “Practical Treatment of Pain in Patients with Chronic Wounds: Pathogenesis Guided Management.” American Journal of Surgery 188(Suppl):31-5, July 2004.
72. World Health Organization. Cancer Pain Relief, 2nd ed., Geneva, Switzerland: Author, 1996.
73. American Geriatrics Society, Panel on Pharmacological Management of Persistent Pain in Older persons. “Pharmacological Management of Persistent Pain in Older Persons.” Journal of American Geriatrics Society 57(8):1331-46, August 2009.
74. Washington State Agency Medical Directors Group. Interagency Guideline on Opioid Dosing for Chronic Non-cancer Pain: An Educational Pilot to Improve Care and Safety with Opioid Treatment. Olympia, WA: Author, 2010.
75. Edlund, M.J., et al. “Risk Factors for Clinically Recognized Opioid Abuse and Dependency among Veterans Using Opioids for Non-cancer Pain.” Pain 129(3):355-62, June 2007.
76. Galloway, K.T., Buckenmaier, C.C., et al. “Multimodal and Multidisciplinary Therapy for Pain Management. Special Report: War on Pain.” American

Nurse Today (Suppl):13-8, September 2011.
77. Dhalla, I.A., Manmdani, M.M., et al. “Prescribing of Opioids Analgesics and Related Mortality Before and After the Introduction of Long Acting Opioid.” Canadian Medical Association Journal 181(12):891-6, December 2009.
78. Comptom, W.M., Volkow, N.D. “Major Increases in Opioid Analgesic Abuse in the United States: Concerns and Strategies.” Drug and Alcohol Dependence 81:103-7, 2006.
79. FDA. Risk Evaluation and Mitigation Strategies (REMS) for Extended-release and Long-acting Opioids. Available at: http://www.fda.govdrugs/drugsafetyinformation/bydrug class/ucm16346.htm. Last accessed July 9, 2014.
80. FDA. New Safety Measures Announced for Extended-release and Long-acting Opioids. Available at: http://www.fda.govdrugs/drugsafetyinformation/bydrugclass/ucm363722.htm. Last accessed April 16, 2014.
81. Osborne, R., et al. “The Pharmacokinetics of Morphine and Morphine Glucuronides in Kidney Failure.” Clinical Pharmacology & Therapeutics 54:158-67, 1993.
82. Twillman, R.K., et al. “Treatment of Painful Skin Ulcer with Topical Opioids.” Journal of Pain and Symptom Management 17(4):288-92, April 1999.

13

Skin—Challenging Conditions

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Objectives

After completing this chapter, you will be able to:

- differentiate between contact allergic dermatitis and contact irritant dermatitis
- define moisture-associated skin damage (MASD)
- describe the prevention and treatment of incontinence-associated dermatitis (IAD)
- describe the International Skin Tear Advisory Panel (ISTAP) classification system for skin tears
- indicate prevention and treatment care options for skin tears
- explain basic management of psoriasis
- describe the topical therapy used for epidermolysis bullosa (EB)

- define the concepts of skin failure, terminal ulcers, and skin changes at the end of life.

Introduction

Differentiating skin conditions can be challenging to nondermatology practitioners. In this chapter, we have selected some skin conditions that clinicians may see in their everyday practice or clinic setting.

Contact Dermatitis

Contact dermatitis is a condition in which the skin becomes red, sore, or inflamed after direct contact with a substance. Contact dermatitis symptoms depend on the cause and how sensitive the person is to the substance. Two types of dermatitis will be discussed in this chapter: contact allergic dermatitis and contact irritant dermatitis.

Contact Allergic Dermatitis

Contact allergic dermatitis must have a sensitizing exposure followed by a repeated exposure to the allergen to elicit the contact allergic reaction. About 80% of contact dermatitis is irritant and can occur on the first exposure due to moisture or another irritant on the wound surface.[1] Allergic reactions are often bright red and itchy and follow the contours of the responsible allergen. The most common wound associated with contact allergy is the venous leg ulcer where the ulcers are often recurrent and chronic.

To identify a contact allergen, patch tests are performed to the North American series of approximately 50 contact allergens.[1] The patches are applied to the back in a well that is occluded with tape at concentrations to identify a contact allergen but not cause an irritant reaction in most individuals tested. The tape is removed at 48 hours; the location of the patches is marked with a skin marker and a final reading is done at 96 hours when irritation from the tape and occlusion disappears and the true allergens are more likely to be identified. Contact allergic dermatitis is a type 4 lymphocytic immune response that causes eczematous changes on the skin surface like a tuberculin test. It is different than the immediate prick test through a drop of an allergen on the forearm that relates to type 1 IgE reactions related to asthma. It is also valuable to apply 1-cm pieces of wound products on normal skin for 48 hours to detect product allergies that may not relate to common allergens.[1]

The most common contact allergens are topical antibiotics (e.g., bacitracin, neomycin), preservatives (propylene glycol, Quaternium-15/other formaldehyde-releasing preservatives), fragrances (including Balsam of Peru), and components in hydrocolloids (colophony, carboxymethylcellulose) or the three classes of topical steroids.

Patients with an allergic reaction may have bright red dermal erythema often with surface vesicles or bullae that are associated with local pruritus and burning sensations. Treatment is with topical steroids and possible additional topical calcineurin inhibitors (tacrolimus, pimecrolimus). Calcineurin is an enzyme that activates T

cells of the immune system. T cells (also called T lymphocytes) are a type of white blood cell that plays a key role in cell-mediated immunity. Because calcineurin inhibitors suppress the immune system, they are known as immunosuppressants.[2] Topical calcineurin inhibitors may be used to treat inflammatory skin conditions such as atopic dermatitis when other treatments have failed. Severe reactions may require oral antihistamines and oral steroids (see Fig. 13-1).

Figure 13-1. Contact allergic dermatitis. (Reprinted with permission from Goodheart, H., Gonzalez, M. *Goodheart's Photoguide to Common Pediatric and Adult Skin Disorders*. Philadelphia, PA: Wolters Kluwer Health/Lippincott Williams & Wilkins, 2015.)

Contact Irritant Dermatitis

Contact irritant dermatitis is the most common type of dermatitis. Contact irritant dermatitis is not a cellular-mediated response and is due to direct contact leading to physical and chemical damage of the skin. It is not caused by an allergy but rather the skin's reaction to irritating substances or friction. Irritating substances may include acids and alkaline materials such as soaps and detergents, fabric softeners, solvents, or other chemicals. Very irritating chemicals may cause a reaction after just a short period of contact. Milder chemicals can also cause a reaction after repeated contact.

Contact dermatitis presents as a red, itchy rash caused by direct contact with a substance or an allergic reaction to it. The rash isn't contagious or life threatening, but it can be very uncomfortable (see Fig. 13-2).

Figure 13-2. Contact irritant dermatitis. (Reprinted with permission from Goodheart, H., Gonzalez, M. *Goodheart's Photoguide to Common Pediatric and Adult Skin Disorders*. Philadelphia, PA: Wolters Kluwer Health/Lippincott Williams & Wilkins, 2015.)

Many of the contact irritants are known as specific reaction patterns: moisture-associated skin damage (MASD) (incontinence-associated skin damage due to stool and/or urine, intertriginous dermatitis (ITD) due to sweat, peristomal-associated skin damage due to stomal effluent, and periwound-associated dermatitis due to wound exudate) and medical adhesive–related skin injury (MARSI).[1] It may also occur from occlusive dressings, bandages, and plastic sheets or pads placed under or around patients. Excess wound exudate may also be a sign of local or deep and surrounding infection.[1]

Identifying and removing the cause is central to resolution of the problem. If you can avoid the offending substance, the rash usually clears up in 2 to 4 weeks. In addition, exudate and other types of moisture can be prevented from contact with the skin with various barrier products: film-forming liquid acrylates, petrolatum, zinc oxide paste, or windowed dressings (hydrocolloids, films). Stool hardeners, sweat inhibitors, or greater care with products next to the skin can also help prevent contact irritant dermatitis.[1]

Moisture-Associated Skin Damage

Just as dry skin (xerosis) can be a problem, exposure to excessive moisture can also cause skin damage. MASD outlines a variety of conditions characterized by inflammation and in more severe stages erosion or denudation of the epidermis resulting from prolonged exposure to various sources of moisture and irritants (e.g., urine, stool, perspiration, wound exudate, and ostomy effluent).[3]

MASD is actually a type of contact irritant dermatitis, and it is to be considered as an umbrella term including four distinct clinical entities: incontinence-associated dermatitis (IAD), ITD, periwound skin damage, and peristomal skin damage.[4] Since June 2019, the World Health Organization (WHO) includes skin conditions caused by bodily fluids in their latest International Classification of Diseases (ICD-11) for Mortality and Morbidity Statistics under the index of "contact irritant dermatitis."[3] Contact irritant dermatitis is defined as an eczematous reaction provoked by acute or prolonged and repeated contact with a substance or substances which are injurious to the skin. Common irritants include defatting agents (solvents, soaps, and detergents), acids (both inorganic and organic), and alkalis (e.g., sodium hydroxide and wet cement). More specifically, MASD is specified by the index term contact irritant dermatitis, due to friction, sweating or contact with bodily fluids. This index term covers following conditions: (a) primary irritant napkin dermatitis, (b) ITD due to friction sweating or contact with body fluids, (c) contact irritant dermatitis due to saliva, (d) contact irritant dermatitis due to incontinence, (e) contact irritant dermatitis related to stoma or fistula, and (f) contact irritant dermatitis related to skin contact with prostheses or surgical appliances[5–13] (see Chapter 21, Tube, Drains, and Fistula Management).

Common causes of MASD include incontinence of stool and/or urine (IAD), wound exudates (see Chapter 6), fistula or stoma effluent (see Chapter 21), and perspiration, mucus, or saliva.[5–8] Skin damage from moisture is distinct from pressure, and differentiating the correct etiology between these two types of skin injuries is not always easy,

but it is important for appropriate treatment and prevention[8–11] (Table 13-1).

Table 13-1 Differential Diagnosis of MASD and Pressure Ulcers in the Perineal and Genital Area

Characteristic	Incontinence-Associated Dermatitis (IAD)	Pressure Ulcer (PrU)
Location	Often in skin folds Diffuse	Usually over bony prominence Well circumscribed
Color	Red or bright red	Red to bluish/purple
Depth	Intact skin to partial-thickness wound	Intact skin to partial- or full-thickness wound
Necrosis	None	May be present
Pain and itching	May be present	May be present

Adapted from Gray, M., Bohacek, L., Weir, D., et al. "Moisture vs Pressure. Making Sense Out of Perineal Wounds." *Journal of Wound, Ostomy, and Continence Nursing* 34(2):134-42, 2007.

The development and severity of MASD depend on a number of intrinsic and extrinsic factors. It is common among individuals with excessive perspiration, increased dermal metabolism (i.e., elevated local temperature), abnormal skin pH, history of atopy (i.e., genetic susceptibility to contaminants/irritants), deep body folds, dermal atrophy, and inadequate sebum production. Extrinsic factors that may precipitate and exacerbate MASD are chemical/biologic irritants, mechanical stress on the skin (e.g., friction, pressure, shear), fungal/candidiasis proliferation, seasonal or environmental factors (e.g., humidity), incontinence (urine, fecal, or both), and hygienic practices.[3,8,14,15] Identification and classification of gluteal and buttock skin conditions and wounds remains a challenge.[12]

Incontinence-Associated Dermatitis

The remainder of this section will focus on MASD caused from incontinence of urine, stool, or both. IAD is sometimes referred to as perineal dermatitis; the term “diaper dermatitis” should no longer be used (Fig. 13-3). IAD is defined as “a form of irritant dermatitis that develops from chronic exposure to urine or liquid stool.”[6,7,9]

Figure 13-3. Incontinence-associated dermatitis (IAD). (Photo courtesy of KDS Consulting.)

The etiology of IAD is complex and related to the combination of chemical and physical irritation. When the skin is exposed to urine and/or stool, the pH raises into the alkaline range, increasing the

hydration of the stratum corneum, the outermost layer of the epidermis causing alterations in lipid rigidity. As a consequence, the permeability of the skin is increased, and the absorption and penetration of potential irritants are enhanced, reducing the barrier function of the skin. Increased skin pH also raises the fecal enzymatic activity and increases the risk of bacterial colonization, which may cause cutaneous infections. Urine diminishes tissue tolerance to friction (besides shear and pressure). Friction increases when the skin moves across clothing, absorptive incontinence devices, bed, or chair surfaces. Excessive care and frequent cleansing of the skin also lead to chemical and physical irritation, which weakens the skin and might aggravate the clinical manifestation. IAD is considered a risk factor for pressure injuries.[16–18]

In a study by Larner and colleagues,[19] no irritation was present on the arms of normal volunteers when exposed to water; however, skin irritation was seen when repeated application of alkaline urine was place on the arms. This led them to suggest that the skin did not have time to recover from repeated exposure to alkaline urine and return to its usual normal acidic pH. Because skin that is wet may have a higher frictional coefficient, it may be more vulnerable to damage from mechanical trauma such as when cleaning or rubbing the skin or when moving/repositioning a person.[20]

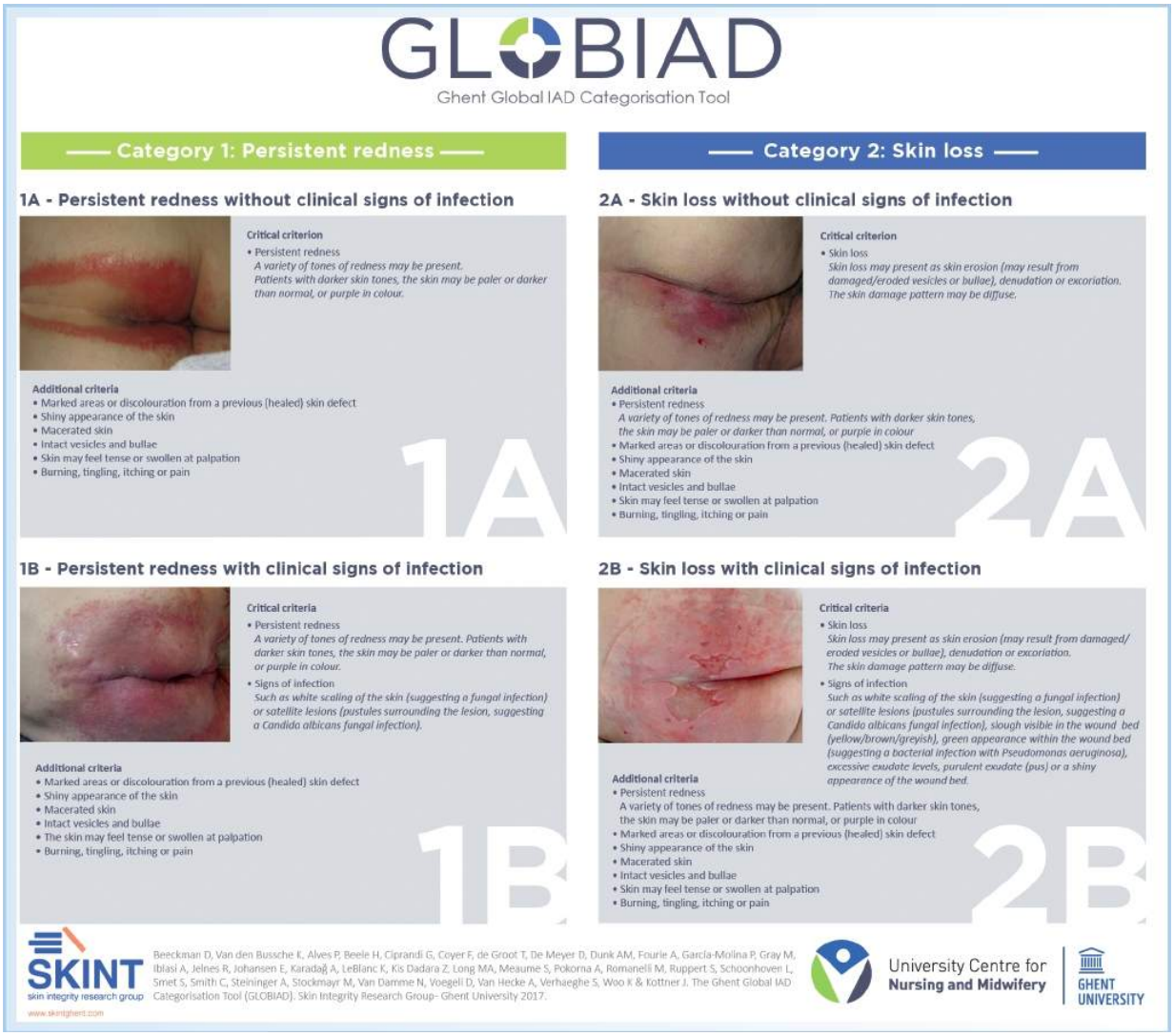
Literature[8–10] has advocated for the use of the term MASD to identify these skin lesions. Gray and Giuliano[18] studied the prevalence of IAD in adult incontinent patients in acute care in 36 states in the United States and found that 2,492 out of 5,342 (46.6%) were incontinent of urine, stool, or both. IAD prevalence rate was 21.3% (1,140/5,342) for the entire sample. However, also half of those with incontinence (1,140/2,492) or 45.7% had IAD. Most had mild (52.3%, 596/1,140) or moderate IAD (27.9%, 318/1,140), with only 9.2% (105/1,140) having severe IAD. Fungal rash was present in 14.8% (169/1,140) persons with IAD,[18] while in a study,[21] fungus was present in 32% (12/38) patients with IAD.

IAD is believed to be reversible, begins as persistent redness, and progresses to partial-thickness skin injury but not full-thickness wounds.[8] Skin assessment classification/categories can be found in

the literature.[22–28] Concerns about the complexity of some of the tools and the amount of time that it can take clinicians to complete have been raised; therefore, they might be better for research studies rather than in everyday clinical practice.[27]

In 2015, a simple approach was recommended by an international expert panel, classifying IAD in three categories based on the level and severity of skin injury.[29] Category 0 was suggested for intact skin without redness in incontinent patients; category 1 indicated mild IAD with erythema (and edema) but intact skin, and category 2 indicated moderate to severe IAD with erythema (and presence of vesicles, bullae, skin erosion, skin denudation, skin infection) and skin breakdown. In 2018, this classification was further developed, and psychometric properties were evaluated by Beeckman et al.[30] resulting in the Ghent Global IAD Categorization Tool (GLOBIAD) (Table 13-2). According to the GLOBIAD, IAD can be categorized in two categories based on the presence of persistent redness (category 1) and skin loss (category 2). Both categories are subdivided into IAD without (category A) and with (category B) clinical signs of infection.[30]

Table 13-2 GLOBIAD



There are many guidelines and recommendation in the literature that can be used to develop best practices for prevention and treatment of IAD.[3,14,15,31]

Contact of the skin with urine and or feces is the primary criterion to develop IAD.[29,32] To minimize contact of the skin with urine and feces, the promotion of continence, the application of absorbent products, and skin cleansing were recommended. Increasing insights concerning the influence of the microclimate (wetness, pH) on the skin barrier structure and functioning have contributed to the optimization of absorbent product design and cleansing procedures.[33]

If possible, prevention of IAD should target reversible causes of incontinence (e.g., urinary tract infection, weak pelvic floor muscles) to promote continence. For this purpose, noninvasive behavioral interventions (e.g., bladder or bowel training program, nutritional and fluid management, mobility enhancement) are mostly sufficient. In some cases (e.g., pelvic organ prolapse), surgery is needed.[33,34]

A plethora of absorbent incontinence products are available, varying from underpads to body-worn products (pads, diapers, and pull-ups). The design of contemporary products has focused on improving the absorption capacity (to avoid overhydration of the stratum corneum) and promoting a slightly acidic pH at the skin surface (to promote stratum corneum integrity and to reduce bacterial overgrowth).[35] The absorption capacity of absorbent products has been increased through the incorporation of superabsorbent polymers which could absorb up to 30 to 50 times their weight and lock the liquid inside.[20] Strategies to optimize the pH of the skin's microclimate included increased breathability and the incorporation of spiral-shaped fibers.[35] A certain degree of breathability of absorbent products is necessary, as occlusive conditions were associated with a rise in humidity, temperature, and pH of the skin's microclimate.[20] An important aspect while using absorbent products is to change these products as frequent as necessary, at least after each episode of fecal incontinence.[4]

Other strategies to prevent contact of the skin with urine and feces are the use of indwelling urinary and fecal catheters and anal pouches. However, no high-quality studies were identified comparing the effectiveness of these products in relation to the use of absorbent products for the prevention of IAD.[29] Furthermore, important side effects were described: patient discomfort, risk of infection, medical device related pressure injuries, and further reduction of continence.[14] Therefore, the use of indwelling devices and anal pouches for the prevention of IAD is recommended by exception and temporarily (e.g., in severe forms of incontinence in high-risk patients).[33]

Skin cleansing aims to remove irritants (urine, feces, debris, microorganisms) from the skin.[33] Traditionally, alkaline soap and water

have been used for skin cleansing. However, this method has been discouraged as the application of alkaline soap changes the protective acid mantel of the skin to an alkaline microclimate. As described above, an alkaline microclimate compromises skin barrier integrity and promotes the colonization of pathogens.[4] Furthermore, the use of water enhances the overhydration of the stratum corneum.[29] To protect the skin barrier, a skin cleansing product with a slightly acidic pH, close to normal skin pH, has been recommended.[15] In addition, the use of soft materials (wash gloves/cloths, towels) is preferred to allow gentle skin cleansing and drying, as a wet skin is more susceptible to friction damage.[15] More skin barrier friendly alternatives instead of the use of (alkaline) soap are bath/shower oil (without perfume or other possible allergens), no-rinse liquid skin cleansers, and premoistened wipes.[4,29] The latter two offer the additional advantage of avoiding the use of water. In addition, manual drying is not always necessary because of the quick-drying properties of these products, which reduces the risk of friction damage.[29]

Furthermore, as the application of no-rinse skin liquid skin cleansers and premoistened wipes no longer requires the use of wash basins, nursing time for skin cleansing decreases, and a potential source of hospital acquired infections is excluded.[4] The active ingredients of skin cleansing products are surfactants which reduce surface tension and facilitate the gentle removal of soilage and dead skin cells. The use of nonionic (uncharged) surfactants is preferred, as these are in general less irritating than anionic (negatively charged) surfactants. Examples of nonionic surfactants are polyethylene glycol (PEG), alkyl polyglycoside (APG), polysorbates, and octoxynols.[29]

Protecting and Enhancing the Skin Barrier

In incontinent patients, contact of the skin with urine and feces cannot be completely excluded by using absorbent products and regular skin cleansing. Furthermore, the presence of an alkaline pH, moisture, and irritants compromises the integrity and function of the lipid lamellae which are embedding the corneocytes.[29] As described before, the lipid lamellae are a vital component of the stratum corneum, impeding the loss and entry of water and the entry of foreign, irritating substances.[29] In addition, incontinent patients with a dry skin are at increased risk for skin irritation because scaling and fissuring promote the penetration of irritants.[3]

To preserve the integrity of the lipid lamellae in the stratum corneum, the application of leave-on products with skin protecting and/or moisturizing properties is recommended.[29] Leave-on products are defined by the cosmetics legislation of the European Commission as cosmetic products that are intended to stay in prolonged contact with the skin, the hair, or the mucous membranes.[29]

Leave-on products with skin protecting properties provide a physical barrier against moisture and irritants from urine and feces. The main types of skin-protective ingredients are petrolatum, zinc oxide, dimethicone, and acrylate terpolymer. It is known that the application of petrolatum and zinc oxide may affect the absorbance capacity of incontinence products.[3,14,29] Leave-on products with moisturizing properties replenish components of the stratum corneum to enhance the skin barrier function and to promote a healthy skin. The active ingredients of this type of leave-on products can be divided into three groups. The first group of ingredients consists of humectants and emollients which aim to treat or ameliorate symptoms of dry skin. Humectants, such as amino acids (e.g., pyrrolidone carboxylic acid, urocanic acid), lactic acid, propylene glycol (glycerine), and urea, replenish the natural moisturizing factor.[3] As these ingredients attract and bind water, they are contraindicated to use in patients with an overhydrated skin or where maceration is

visible.[29] The application of emollients (e.g., plant oils, petrolatum) reinforces the sebum layer at the skin surface to soften and smoothen the skin. These ingredients act by occlusion and by filling in the crevices between the corneocytes.[29] The second group of ingredients consists of lipids similar to those found in the stratum corneum lipid lamellae (e.g., ceramides). The third group represents other components present in a healthy skin, such as essential fatty acids (e.g., linoleic acid), vitamins, and antioxidants.[3]

Most leave-on products contain either ingredients with skin protecting properties, as moisturizing properties. The actual functioning of leave-on products depends on the combination of the ingredients, the overall formulation, and the amount and frequency of application.[3,33] Leave-on products can also be incorporated into liquid skin cleansers or premoistened disposable wipes to allow the performance of skin cleansing, skin moisturizing, and skin protecting in one or two steps.[36] Especially single-step interventions aim to promote efficiency and staff adherence.[14]

Treatment of IAD

Available guidelines and reviews do not provide strict distinctions between interventions for the prevention and treatment of IAD.[3,29,31] Based on the primary characteristics of IAD, treatment should focus on the promotion of healing and reepithelialization, the reduction of skin inflammation, and the eradication of cutaneous infections.[29] In patients with IAD, irritants and microorganisms may more easily penetrate the epidermis due to reduction of the stratum corneum integrity.[3,29] To create a healing environment, the key principles of IAD prevention should at least be applied:

1. Minimize contact of the skin with urine and feces (incontinence management, skin cleansing).
2. Protect and enhance the skin barrier (use of leave-on products).

Leave-on products can be used to treat mild contact irritant dermatitis (e.g., IAD category I).[37] However, some products are not suitable for use on damaged skin (IAD category II). For open, exudating lesions, lotions with wound desiccating properties are preferred above barrier creams.[4] If the skin condition does not improve after 3 to 5 days of treatment, or an infection is suspected, specialist advise should be sought.[29] In case of severe inflammation, treatment with steroid-based, topical anti-inflammatory products may be prescribed by a physician and/or nurse practitioner. However, because of possible side effects (e.g., thinning of the epidermis), these products should not be used for routine treatment of IAD.[14]

Secondary skin infections in patients with incontinence are mostly caused by *Candida albicans*, originating from the gastrointestinal tract.[29,38] However, other microorganisms, such as *Escherichia coli* (present in feces), *Staphylococcus aureus* (present at the skin), and *Pseudomonas aeruginosa* (present in the environment), may also cause infection of an IAD lesion.[18] Therefore, to ensure organism-specific treatment, microbiological samples should be collected before applying any topical antimicrobial product (e.g., antifungal cream, topical antibiotic, anti-inflammatory product). In general, topical

antimicrobial products should not be used as a routine, to prevent the development of antimicrobial resistance.[29,31] If the use of dressings is necessary to promote healing in severe IAD lesions, atraumatic tapes or adhesives should be used to prevent skin damage and pain during dressing changes.[3]

As described earlier, a wet skin has a higher coefficient of friction.[39] In addition, in patients with incontinence, urine and feces tend to accumulate between the dressing and the skin.[40] Therefore, dressings should be used temporarily and changed regularly to avoid prolonged contact of the skin with soiled and saturated dressings.[3]

To help shorten the time to implement what is known in the literature about IAD, the ACT© mnemonic (see Table 13-3) may be a useful tool for knowledge translation into everyday practice. ACT stands for assess, cleanse, and treat.[14] Table 13-3 provides a succinct summary of different skin care products that can be used to protect and treat skin from IAD.[4]

Table 13-3 ACT© Approach to Preventing and Treating the Cause of IAD

Assess	**Assess skin at least daily and following each episode of incontinence; include the following in your skin assessment:** • ***Cause of skin change****: Is it urine, stool, or both; what is the length of time skin is exposed to moisture?* • ***Color of skin:*** *Is it brighter or darker than usual skin tone; are there signs of inflammation?* • ***Condition of the skin:*** *Is the skin closed/intact or open/eroded skin; Are there any blisters, rashes, infection, fungus, or satellite lesions?*
Cleanse	**Cleanse skin:** • **Use surfactants, pH-balanced skin cleansers** • **Maintain acidic pH by avoiding alkaline products** • **Reduce friction/avoid rubbing** **Correct cause of IAD (if possible)** **Contain incontinence of urine and/or stool: use internal or external devices or body-worn absorbent garments**
Treat	**Treat vulnerable skin proactively with:** • **Silicone-based barrier ointments or creams** • **Petrolatum or zinc oxide ointments or creams** • **Film-forming liquid acrylates (cyanoacrylate formulations)—sprays, wipes (roll on, stick, pad)** **Treat damaged skin:** • **Wick skin moisture away with superabsorbents** **Traumatic injury:** • **Avoid tapes that can damage skin** • **Use fluid lock dressings**

ACT, assess, cleanse, treat; IAD, incontinence associated dermatitis.
As published in McNichol, L.L., Ayello, E.A., Phearman, L.A., et al. Incontinence-Associated Dermatitis: State of the Science and Knowledge Translation. *Advances in Skin and Wound Care* 31(11):502-13, 2018. © Ayello & Sibbald 2017.

Consistently implemented IAD prevention strategies have resulted in a decrease prevalence of IAD from 60% to 16% in the pediatric population.[41] Gray et al.[9] summarized several research studies about the prevention of IAD and concluded that a routine perineal skin protocols that avoided soap and used cleansing products with a pH range of normal skin were effective along with reducing skin scrubbing and friction. Use of products that moisturize and protect the skin was recommended.[9] Holistic care also requires interventions to address and minimize the episodes of incontinence such as a scheduled toileting program. Other strategies to prevent skin exposure to urine or stool include use of containment devices such as condom catheter, anal pouch, or bowel management systems. If absorptive products are used, such as some of the newer body-worn garments, make sure that they wick urine or stool away from the skin.[18,29,42,43] Bliss and colleagues[43] found that skin pH was more acidic when a spiral-

shaped fiber incontinence brief was worn compared with a standard brief. Some of the recommendations for a structured skin care regimen include cleaning the skin daily and after each incontinence episode using a no-rinse cleanser; not scrubbing the skin; applying humectants or emollient moisturizer; barrier creams; ointments with petroleum, zinc oxide, or dimethicone; or skin sealant products (see Table 13-4).[9–11] If a fungal infection is also present, this will require additional antifungal products.[9,11]

Table 13-4 Clinical Barrier Options for Protection Against Moisture-Associated Skin Damage

Product Category	Advantages	Disadvantages
Petrolatum-based barrier ointments (a soft paraffin or wax mixture)	• Forms a seal over the skin to reduce transepidermal water loss; optimal effect when applied to slightly dampened skin • Thickening properties to protect against mechanical damage and serve as a physical barrier against irritants	• Petrolatum may melt under heat • May leave a greasy residue that interferes with primary dressing adherence and absorption • May build up in the pores and attract dirt and bacteria, increasing the risk of folliculitis
Zinc oxide–thickened petrolatum-containing barrier ointments	• Repels irritants in exudate, urine, and other fluids • Thickening properties to protect against mechanical damage • Anti-inflammatory and antioxidant	• Preparations may be thick and difficult to apply and remove • May interfere with primary dressing adherence and absorption • Ointment may inadvertently get into the wound and interfere with antimicrobial ingredients
Silicone-based barrier ointments (such as dimethicone)	• Dimethicone is permeable to water vapor that allows evaporation of perspiration and minimizes the risk of miliaria (heat rash) • Conformable to periwound area or area of at risk skin • Easy to spread and feels less greasy on skin	• Thick ointments may interfere with primary dressing adherence and absorption • Ointment may inadvertently get into the wound and may not be indicated for use in open wounds
Film-forming polymers in water or organic solvents	• Forms a mechanical barrier over periwound surface • Thin and nonmessy • Allows for adherence of wound dressing and protects against skin stripping during dressing changes	• Certain organic solvents may cause stinging and irritation; also obvious inhalation and fire hazards • Much of the applied liquid evaporates, leaving a very thin, insubstantial film compared with cyanoacrylates
Cyanoacrylate formulations	• Creates a moisture-resistant barrier on skin • Protects against friction-induced skin damage • Does not require solvent, so all of the applied liquid turns into a barrier • Bonds to skin via polymerization in situ so very resistant to wash off or premature removal	• Cyanoacrylates are somewhat expensive raw materials in skin barrier formulations • Individuals may be allergic to cyanoacrylates

Intertriginous Dermatitis

Another type of MASD is or ITD. It is “the term for skin damage caused by trapped perspiration and frictional forces between opposing skin surface.”[12] The prevalence/incidence of ITD is unknown, and there is no formal risk assessment tool.[44] Clinicians should be looking in skin folds including the inframammary, axillary, and inguinal skin folds as well as the abdominal or pubic panniculi[7] (Fig. 13-4). Since skin-to-skin moisture and friction have been hypothesized as the cause of ITD, a high-risk population for ITD is the bariatric and obese patient population. Others at risk include women with large pendulous breasts, persons with stocky necks, and children with skin folds.

Figure 13-4. Intertriginous dermatitis (ITD). Intertrigo between the skin folds of the breast of a female patient with a body mass index of 60. Note the fissure at the base of the skin fold. (© Baranoski and Ayello, used with permission.)

As moisture accumulates in a skin fold, the potential for growth of microorganisms exists.[45] As skin pH becomes more alkaline, Black et al.[7] report that ITD cultures have shown growth of the following organisms: *S. aureus*, group Aβ-hemolytic streptococcus, *Pseudomonas*, *Proteus mirabilis*, *Proteus vulgaris*, enterococci, vancomycin-resistant enterococci, and fungus such as *Candida*.

Careful assessment of skin folds for ITD is important. This should include under the breasts (usually if pendulous), abdomen, knees, and elbows. Also look in the neck region, on the back below the scapulae (sometimes called “angel wings”) and in the fourth and fifth toe webs as intertrigo spreads proximally.[44] Typically, ITD begins as mild, mirror-image erythema of the skin.[7] With continued exposure to the moisture, the skin becomes more inflamed and macerated. Skin erosions, oozing, crusting, itching, pain, and odor may be present.[7] Efforts to prevent ITD focus on appropriate skin hygiene including keeping the skin clean and dry, using skin pH neutral products, avoiding abrasive and friction using skin care, and “pat” drying the skin.[44] Other ITD management principles can be found in (Table 13-5). Treatment includes continuing the skin care regimens for cleansing as well as soaks with various astringent solutions, specialized dressings designed to be placed in skin folds to absorb moisture, and if candidiasis is present to use antifungal powders.[7]

Table 13-5 Intertriginous Dermatitis (ITD)-Management Principles

- Minimize skin-on-skin contact and friction
- Remove irritants from the skin and protect skin from additional exposure to irritants
- Wick moisture away from affected and at-risk skin
- Control or divert the moisture source
- Prevent/treat secondary infections

Based on Sibbald, R.G., Kelley, J., Kennedy-Evans, L.K., et al. A Practical Approach to the Prevention and Management of Intertrigo, or Moisture-Associated Skin Damage, Due to Perspiration: Expert Consensus on Best Practice. *Wound Care Canada* 11(2), 2013.

Skin Tears

The International Skin Tear Advisory Panel (ISTAP) defines skin tears as “A traumatic wound caused by mechanical forces, including removal of adhesives. Severity may vary by depth (not extending through the subcutaneous layer).”[46] Individuals at the extremes of age and the chronically or acutely ill have an increased skin tear risk.[47,48] An aging population coupled with an increased prevalence of chronic diseases translate into longer healing times for the majority of wounds, including skin tears.[49] Individuals suffering from skin tears complain of increased pain, which in addition to other biopsychosocial factors associated with chronic wounds, such as physical disability, social needs, and mental anguish, may negatively impact an individual’s quality of life.[50,51]

Healthcare professionals must become cognizant of which individuals are at risk for developing skin tears, how to prevent these wounds, and how to treat them once they occur. In recent literature, there has been increasing attention given to skin tears, but there has been no gold standard developed for their management. While the prevention of skin tears is the primary focus, healthcare professionals must be equipped to manage these wounds when they do occur. By recognizing which patients are at risk for skin tears, preventing skin injuries, and using appropriate nonadherent dressings, can save patients undue pain and suffering.[47,52,53]

There has historically been limited interest in skin tears, as demonstrated by the paucity of quality publications addressing these wounds. There exists a perception among healthcare professionals that skin tears are minor wounds, not requiring focused attention, resulting in an inadequate scientific study devoted to these wounds.[48] In an effort to redirect awareness toward this largely unheeded healthcare issue, ISTAP, consisting of 12 internationally recognized key opinion leaders, was established. Key consensus statements were produced based on current knowledge and evidence focusing on the epidemiology, etiology, pathophysiology, assessment, prevention, treatment, and documentation of skin tears.[47] Subsequent work by

ISTAP also included the development and validation of a skin tear classification system and a comprehensive tool kit to assist clinicians with the development of a broad prevention program.[52,54]

Content validity of the ISTAP classification system including the newly developed “skin flap” definition was established by a panel of 17 international experts. After a two-round Delphi process, consensus was achieved about the definition for the concept of a “skin flap” in skin tears. The development of such a definition for the area of skin tears is important because this concept may be interpreted differently depending on one’s educational background.[55] In the field of reconstructive surgery, for example, a “skin flap” is considered a mass of tissue intentionally detached from its original place to be used for grafting for wound repair and organ reconstruction.[56] A clear, internationally accepted definition of a “skin flap” associated with the condition of a skin tear should help to eliminate confusion and to facilitate best practice.[46] Best practice recommendations for the prevention and management of skin tears in aged skin[46] are available to download from www.woundsinternational.com.

“A flap in skin tears is defined[57,58] as a portion of the skin (epidermis/dermis) that is unintentionally separated (partially or fully) from its original place due to shear, friction, and/or blunt force. This concept is not to be confused with tissue that is intentionally detached from its place of origin for therapeutic use, for example, surgical skin grafting.” The three categories of the initial ISTAP tool have remained unchanged. The ISTAP classification system including the newly developed “skin flap” definition is presented in Figure 13-5.

A

B

C

Figure 13-5. ISTAP skin tear classification. A flap in skin tears is defined[57] as a portion of the skin (epidermis/dermis) that is unintentionally separated (partially or fully) from its original place due to shear, friction, and/or blunt force. **(A, B)** Type 1, no skin loss. Linear or flap tear that can be repositioned to cover the wound bed. **(C)** Type 2, partial flap loss. Partial

flap loss that cannot be repositioned to cover the wound bed. **(D)** Type 3, total flap loss. Exposing entire wound bed. (Copyright ISTAP 2019.)

Prevalence

Skin tears are perceived to be common in the extremes of age and critically ill. Although they are seen in practice, the prevalence of skin tears may be underreported, especially in the community.[59] Some researchers have hypothesized that the prevalence rates of skin tears are equal to or greater than those of pressure ulcers.[60–62] Despite these assumptions, limited prevalence studies have been conducted to support this hypothesis. In the LTC setting, the prevalence of STs has been reported to be between 3.9% and 26.0%.[63–66] These findings support the assumptions that the prevalence rates of skin tears, particularly in the LTC setting, closely resemble those of pressure ulcers.[59,67]

When skin tears are reported, the causative factor is often not known. When the cause is known, skin tears are frequently linked to wheelchair injuries, blunt trauma from accidently bumping into objects, transfers, or falls.[48,68,69] Studies[69,70] have shown that the key times skin tears occur are during the peak activity hours of 6:00 AM to 11:00 AM and 3:00 PM to 9:00 PM. In the elderly population, skin tears are often related to the environment.[71] In 1990, Payne and Martin[72] conducted a 3-month, descriptive study in 10 long-term care facilities to describe skin tears, identify risk factors, and determine the rate of healing of skin tears. Among the predominant risk factors, impaired activity, mobility, sensation, and cognition all demonstrated an increased risk for skin tear development. McGough-Csarny and Kopac[73] conducted a similar study in a Veterans Affairs nursing home and concluded that dependency in activities of daily living (ADLs), sensory loss, limited mobility, use of assistive devices, and impaired cognition were risk factors for skin tear development. Patients who are dependent on others for total care are at the greatest risk for skin tears.[70] Dependent patients frequently acquire skin tears during

routine activities, such as dressing, bathing, repositioning, and transferring. Independent ambulatory patients are at the second highest risk, and the majority of their skin tears occur on their lower extremities.[70,74] In the 2011 survey conducted by LeBlanc and Baranoski et al.,[47,48] causes of skin tears included equipment injury, patient transfers, falls, ADL, and treatment and dressing removal. LeBlanc[75] reported that there was sufficient evidence to support that having history of a ST, skin changes associated with aging, having comorbidities, requiring assistance with ADLs, and displaying aggressive behavior increased ST risk; however, other factors including advanced age, gender, polypharmacy, lower extremity edema, nutritional deficits, auditory or visual impairment, cognitive impairment, increased pressure injury risk, and xerosis all require further exploration to establish if they increase ST risk in the LTC population.[75]

Kaya and Saurat[76] hypothesized that the hands, arms, and lower legs were anatomical locations which experienced a higher degree of age-related skin changes as the result of frequent sun exposure. Supporting this claim, LeBlanc[75] reported that 42.4% of STs occurred on the upper extremities and 51.6% on the lower extremities. Koyano et al.[63] hypothesized that potential reasons for the differences in ST prevalence between countries could be related to cultural differences such as the influence culture may have on a society's sun exposure habits. This concept is supported by the belief that anatomical locations having greater sun exposure display more severe photodamage and subsequent skin changes associated with aging and experience more STs.[76]

Although nearly 70% to 80% of skin tears occur on the arms and hands,[68,77] these wounds may occur on other areas of the body as well. Skin tears that occur on the back and buttocks are commonly mistaken for stage 2 pressure injuries. Pressure may be a related cause in skin tears, but the etiology of skin tears differs from that of pressure injuries.[47,52] Skin tears need to be documented as separate occurrences and not grouped into pressure injury categories (see Chapter 14, Pressure Injuries).

Risk Factors

The ISTAP developed a risk assessment pathway[52] (Fig. 13-6) to aid in the prediction of skin tears. The pathway is complemented by the ISTAP risk reduction program (Table 13-6), which will be discussed later in this chapter.

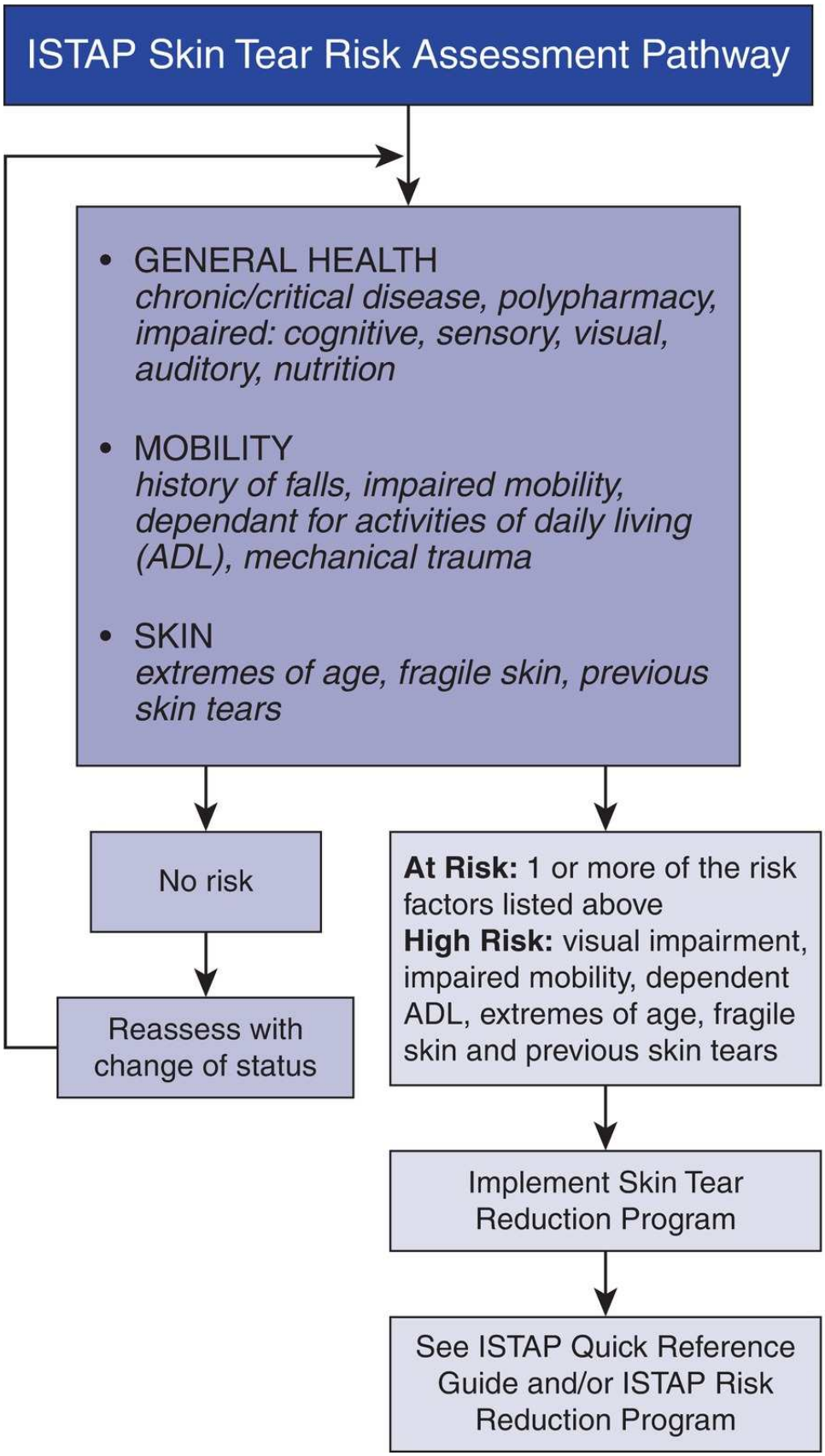

ISTAP Skin Tear Risk Assessment Pathway
• GENERAL HEALTH
chronic/critical disease, polypharmacy, impaired: cognitive, sensory, visual, auditory, nutrition
• MOBILITY
history of falls, impaired mobility, dependant for activities of daily living (ADL), mechanical trauma
• SKIN
extremes of age, fragile skin, previous skin tears
No risk
Reassess with change of status
At Risk: 1 or more of the risk factors listed above
High Risk: visual impairment, impaired mobility, dependent ADL, extremes of age, fragile skin and previous skin tears
Implement Skin Tear Reduction Program
See ISTAP Quick Reference Guide and/or ISTAP Risk Reduction Program

Figure 13-6. Skin tear risk assessment pathway.

Table 13-6 Quick Reference Guide: ISTAP Risk Reduction Program

Risk Factor	Individual	Care Giver/Provider
General health	• Educate the patient on skin tear prevention and promote active involvement in treatment decisions (if cognitive function not impaired) • Optimize nutrition and hydration	• Safe patient environment • Educate the client +/circle of care/caregivers • Protect from self-harm • Dietary consult if indicated • Extra caution with extremes of BMI (<20 or >30) • Review polypharmacy for medications reduction/ optimization
Mobility	• Encourage active involvement if physical function not impaired • Appropriate selection and use of assistive devices	• Daily skin assessment and monitor for skin tears • Ensure safe patient handling techniques/equipment and environment (trauma, activities of daily living [ADLs], self-injury) • Proper transferring/repositioning • Initiate fall prevention program • Remove clutter • Ensure proper lighting • Pad equipment/furniture (bed rails, wheelchair, etc.) • Avoid sharp fingernails/jewelry when having patient contact
Skin	• Awareness of medication-induced skin fragility (e.g., topical and systemic steroids) • Wear protective clothing (shin guards, long sleeves, etc.) • Moisturize skin (lubrication and hydration) • Keep fingernails short	• Individualize skin hygiene (warm, tepid not hot water, soapless or pH-neutral cleaners, moisturize skin) • Avoid strong adhesives, dressings, tapes • Avoid sharp fingernails/jewelry with patient contact
Healthcare setting		
• Implement comprehensive skin tear reduction program • Include skin tears in audit programs • Utilize validated classification system • Develop consultative team (wound care/dietary specialists, rehab/pharmacists)		

General Health

Altered Sensory, Auditory, and Visual Status

Chronic disease, often experienced by the aging population, can lead to altered sensory, visual, auditory, and neuropathic status. In addition, the critically ill and individuals with extremes of age (the very young or the very old) can also be afflicted with altered sensory status.[47] Aging skin puts individuals at risk for skin injury including acute traumatic wounds or skin tears. Care of the geriatric patient often involves managing persons who are among the oldest and fastest-growing segment of the population (85 years and older). As individuals age, the normal wound healing process also slows and the risk of dementia, diabetes, and vascular disease increases. With the increase in the aging population, more individuals will be at risk for developing skin injuries including chronic wounds and skin tears.[78,79]

Altered sensory, visual, auditory, and neuropathic status all contribute to increased fall risks.[78,79] History of falls has been strongly linked in the literature to an increased risk of skin tears.[48,64,71,74,78]

The critically ill, pediatric, and premature neonatal population can also be at an increased risk for skin tear development if alterations in sensory, visual, auditory, or neuropathic are experienced. Care should be taken to ensure that the individual and all those involved in their care are aware of the increased risk of skin tear and ensure protection from potential risk factors.[79,80]

Cognitive Impairment

Despite the paucity in the literature surrounding the causative factors of skin tears, there is evidence to suggest that altered levels of cognition in the elderly[54] pediatric,[80] and critically or chronically ill individual[47] increase the risk of skin tear development.

Various types of dementia and other chronic illnesses lead to altered cognitive status and can be an added challenge to caregivers. Cognitive impairment can lead to decreased adherence to prevention

programs due to lack of comprehension. Aggressive behavior and agitation associated with altered cognition and dementia can also increase the risk of blunt trauma and self-injury resulting in skin tears.[81]

The very young or developmentally challenged individual cannot be forgotten when managing skin tears. Lack of cognition or insight can potentially increase the risk of skin tears in this population.[79]

Nutritional Concerns

Adequate nutrition and hydration help maintain tissue viability.[82] Assessment of each individual's nutritional status is vital to insure health and well-being. The elderly can be at great risk for nutritional impairment. Of the hospitalized elderly, 25% to 30% are undernourished, while 46% to 61% are at risk for being undernourished.[82]

Dehydration can be assessed by monitoring intake, urine output, skin turgor, changes in weight, and lab values such as sodium and serum osmolality. With issues such as diarrhea, vomiting, increased body temperature, or heavily draining wounds, more fluid intake is needed to replenish lost fluid and electrolytes. Adequate hydration is essential in maintaining skin integrity. It can make a difference between a bump causing a bruise and a skin tear.[83]

Polypharmacy

Polypharmacy (i.e., the use of multiple medications that might predispose patients to drug interactions/reactions or confusion) is common among the elderly. Numerous medications can be associated with negative health outcomes (i.e., cutaneous reactions, falls), but more research is needed to further delineate the consequences associated with multiple drug use, especially in elderly patients.[84]

A variety of medications can affect the skin, the most common being corticosteroids, which can interfere with epidermal regeneration and collagen synthesis.[81,83] Steroids may be a contributing factor to skin tear development. Polypharmacy has also been indicated to be an independent risk factor for falls.[84,85] Pervin's[84] research concluded

that individuals receiving four or more medications are at a greater fall risk. In a recent consensus document pertaining to the prevention, assessment, and treatment of skin tears, LeBlanc et al.[47] identified falls as a major risk factor in the development of skin tears as well.

Mobility

Mobility-Related Issues

When skin tears are reported, the causative factor is often not known.[47,72] Nevertheless, skin tears are frequently linked to wheelchair injuries, falls, transfers, or blunt trauma from bumping into objects.[53]

Assistance with Activities of Daily Living

Individuals frequently acquire skin tears during routine activities of dressing, bathing, positioning, and transferring, and those who are dependent on others for total care are at the greatest risk for skin tears.[74,86] Individuals who are independent in ambulation report high numbers of skin tear occurring primarily on the lower extremities.[87–89] A 2011 survey reported that the perceived top causes of skin tears included equipment injury, patient transfers, falls, ADL, treatment, and dressing removal.[48]

History or Risk of Falls

Falls pose a serious risk of skin tears for the elderly. An average nursing home with 100 beds reports 100 to 200 falls annually.[90] Predisposing factors for falls include unsteady gait and balance, weak muscles, poor vision, medications, and dementia. In addition, other factors, such as poor lighting, loose rugs, poorly fitting shoes, floor clutter, continence urgency, and beds or toilets without handrails, also may cause falls.[91] Furthermore, medical conditions such as low blood pressure, stroke, Parkinson's disease, arthritis, Ménière's disease (affects the middle ear—causes vertigo), poorly controlled diabetes, poorly controlled epilepsy, brain disorders, and thyroid problem increase the elderly client's risk for falls.[91] Falls have been associated with contributing to the development of skin tears in the elderly and compromised populations.[48]

Mechanical Trauma (Not Related to Mobility Aids)

Intrinsic factors, such as age, pertain to an individual's inherent biologic or genetic makeup. Extremes in age impact not only on how individuals heal but also on their susceptibility to developing a wound.[53] With increasing age, individuals experience dermal and subcutaneous tissue loss, epidermal thinning, and serum composition changes, all of which cause decreased skin surface moisture.[83] In turn, the skin's elasticity and tensile strength decreases. In the neonatal population, mechanical trauma is the number one cause of skin tears.[79]

Skin Changes Related to Extremes of Age and Critically Ill

Skin tears commonly occur on individuals at the extremes of age (elderly to neonate), the critically ill or medically compromised, and to those requiring assistance with personal care. As the skin ages, there are many changes that occur within the dermis making the skin more susceptible to skin tears as previously addressed.

Prevention

Little has been written about the prevention of skin tears. Following tried and true guideline, recommendation can help prevent skin tears. Best practice guidelines and the ISTAP risk reduction prevention program, gleaned from the literature, may prevent many skin tears.[47,48,52–54]

- Encourage your colleagues and the patient's family members to use proper positioning, turning, lifting, and transferring.
- Promote the use of long sleeves and pants to add a layer of protection.
- Secure padding to bed rails, wheelchair arm and leg supports, and any other equipment that may be used.
- Use nonadherent dressings on frail skin. Always remove these products gently to prevent skin injury.
- Use skin sealants, liquid bandage, or soft silicone or foam dressings to protect vulnerable skin from adhering tapes and dressings.
- Use stockinettes, gauze wrap, or a similar type of wrap to secure dressings and drains rather than tape.
- Use pillows and blankets to support dangling arms and legs.
- Minimize the use of soap and alcohol solvents; consider the use of no-rinse, waterless, or liquid gel cleansers.
- Avoid scrubbing skin when bathing; pat skin dry rather than rubbing it dry.
- Apply a moisturizing or emollient agent to dry skin.
- Provide a well-lit environment to prevent falls.

- Educate staff on the importance of gentle care.

Research[71] has shown that skin tears can be reduced in nursing homes when skin care protocols are used.[52,87] Skin tears in an LTC facility declined from 23.5% to 3.5% with the implementation of a no-rinse, one-step, bed bath protocol rather than soap and water.[92] Skin tears were significantly decreased in two different rural nursing homes when skin care protocols were introduced. Hanson et al. found that skin tear prevalence was reduced from 6.3% to 1.4% in nursing home A and 6.4% to 3.3% in nursing home B.[68] By educating staff and implementing the above-mentioned skin care protocols, skin tears were reduced from a monthly average of 18 to 11.[87] Another study emphasizes the importance of educating nurses and nursing assistants; in a 10-month descriptive study with 30 patients with Alzheimer's disease, 26 of the 30 patients remained free of skin tears using a preventive skin program.[93,94] A study of 416 RNs in two affiliated hospitals demonstrated increased ability to identify and assess skin tears, differentiation of skin tear categories, and increased knowledge of treatment by using a web-based educational program.[95]

Skin Tear Classification System

The initial classification system for skin tears evolved in the late 1980s from the work of Regina Payne and Marie Martin. Their pilot research study led to the development of the Payne-Martin classification system for skin tears.[72,77] The Skin Tear Audit Research Tool (STAR) a modified version of the Payne-Martin system was developed by a coordinated group of Australian national wound care experts and is composed of five types related to amount of skin loss and presence or absence of dusky color of the skin flap. The STAR classification system displays a high level of complexity and subjectivity, and the clinician must determine the percentage of tissue loss combined with the presence of absence of a hematoma and/or dusky colored skin flap, which is highly subjective and potentially too complex for clinicians to use in practice.[54,69]

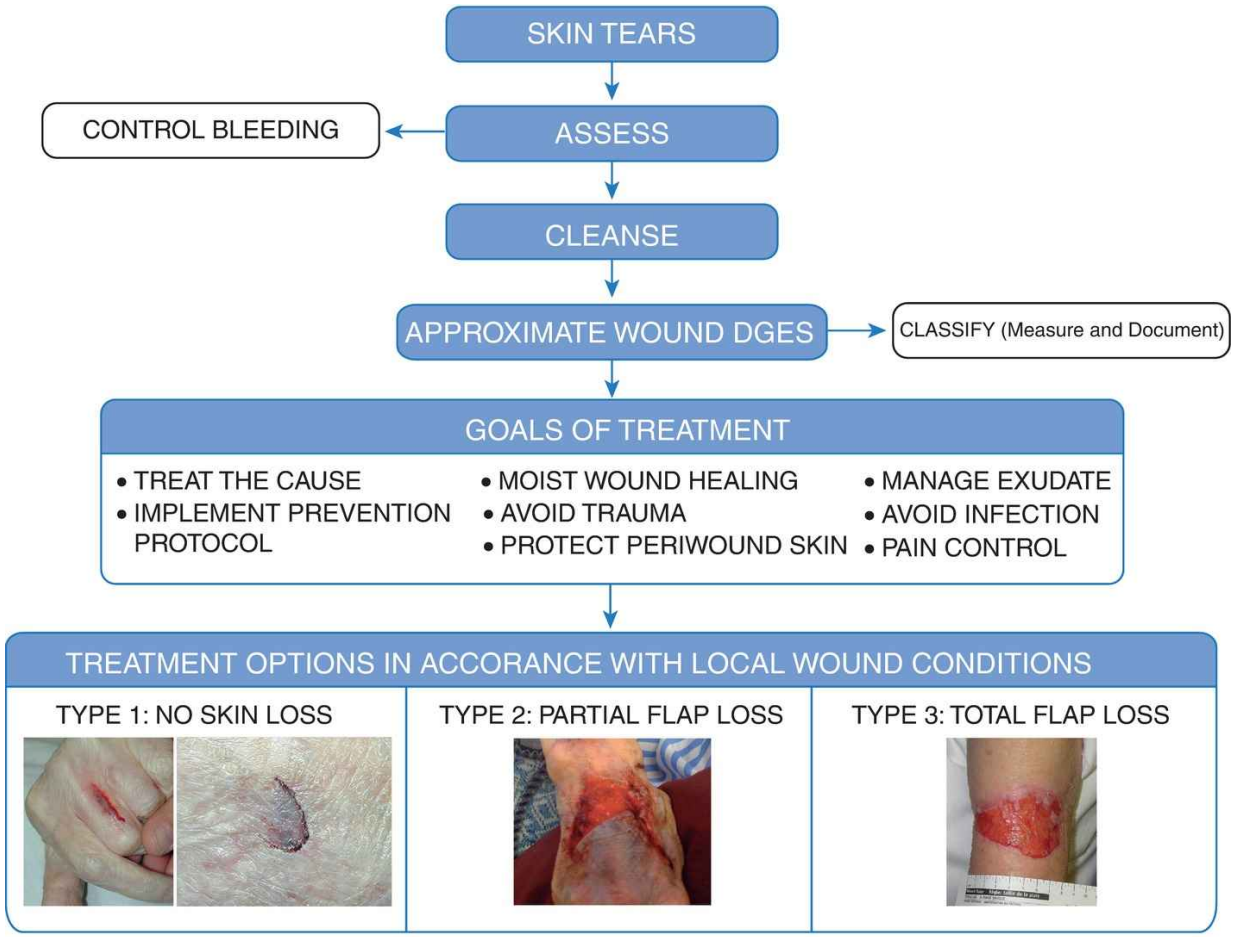
An international cross-sectional survey conducted by LeBlanc, Baranoski, and Regan found that both the Payne-Martin and the STAR classification systems were not widely adopted into global practice and that clinicians overwhelmingly supported a simplified classification system for skin tears.[48] In response to these findings, the ISTAP developed and validated the ISTAP skin tear classification system[54] (see Fig. 13-5). This system is user friendly and can be used by all healthcare providers to document skin tears. The system has been globally disseminated, being translated and validated into various languages.[96–98]

Patient Teaching

Alert the patient/family/caregiver that skin tears often occur over areas of senile purpura in the elderly.

Skin Tear Management

The management or treatment of skin tears varies according to institution and clinician practices. Skin tear management includes assessing the insult, controlling bleeding, wound cleansing, removing any necrotic tissue/debris, realigning the wound edge/flap or pedicle, and classifying, measuring, and documenting the type of skin tear (type 1, 2, or 3). The basic goal of treatment is to treat the cause, avoid further trauma, avoid infection, control pain, manage exudates, and use a moist wound therapy, nonadherent wound dressing (Fig. 13-7).



A flap in skin tears is defined as a portion of the skin (epidermis/dermis) that is unintentionally separated (partially or fully) from its original place due to shear, friction, and/or blunt force.©ISTAP

Figure 13-7. Skin tear algorithm.

Practice Point

Tetanus is an acute, often fatal disease caused by wound contamination with *Clostridium tetani*. Human tetanus immunoglobulin (TIG) neutralizes circulating tetanospasmin and toxin in the wound but not toxin that is already fixed in the nervous system. It should be given according to individual institutional policy, to individuals with interruption of the skin integrity by a nonsurgical mechanism who have not received a tetanus toxoid (Td) inoculation in the past 10 years. The TIG should be given before wound debridement because exotoxin may be released during wound manipulation.[85]

Psoriasis

Psoriasis is a chronic inflammatory skin disease with a reported prevalence of 1% to 3% in Europe and United States.[99,100] It is a noncontagious skin disease that results from an atypical autoimmune response which can lead to accelerated skin growth (hyperproliferation) and dermal inflammation. The etiology of psoriasis is unknown, but genetic factors play a role.[101] Psoriasis can begin at any age but has two peak periods of onset: 15 to 20 and 55 to 60 years of age.[100] The younger the onset, along with a strong family history, the more severe the disease.[102]

The diagnosis of psoriasis is made by obtaining a through history and physical especially noting any hereditary factors. The diagnosis is usually clinical. If needed, tissue samples can be examined under the microscope to look for a flattening and compression of cells (acanthosis). The physical examination should include an examination of the primary lesion and other common areas affected including the scalp.[100] Examination should include the nails and joints as well. Diagnosis can be supported by Auspitz's sign or Koebner's phenomenon.

Auspitz's sign is the appearance of punctate bleeding spots when psoriasis scales are scraped off. This happens because there is thinning of the epidermal layer overlying the tips of the dermal papillae and the blood vessels within the papillae are dilated and tortuous, which bleed readily when the scale is removed[103] (see Fig. 13-8).

Figure 13-8. Auspitz's sign. Silvery-white psoriasiform, well-demarcated plaque with overlapping scale on the elbow and pinpoint sites of bleeding (Auspitz's sign), where the scale has peeled off. (Reprinted with permission from Hall, J.C., Hall, B.J. *Sauer's Manual of Skin Diseases*. Philadelphia, PA: Wolters Kluwer Health/Lippincott Williams & Wilkins, 2017.)

Koebner's phenomenon is a medical term used to describe a specific type of skin reaction among those with psoriasis or other skin disorders. This phenomenon is characterized by the development of new psoriasis lesions at the site of a minor skin injury such as a scrape or insect bite. Common triggers for the appearance of the Koebner's phenomenon include poison ivy, eczema, and minor abrasions[104] (see Fig. 13-9).

Figure 13-9. Koebner's phenomena. Development of new psoriasis lesions at the site of a minor skin injury such as a scrape or insect bite. (Reprinted with permission from Herbert, P., Goodheart, M.D. *Goodheart's Same-Site Differential Diagnosis: A Rapid Method of Diagnosing and Treating Common Skin Disorders*. Philadelphia, PA: Wolters Kluwer Health/Lippincott Williams & Wilkins, 2010.)

Psoriasis is a systemic disease associated with a number of comorbidities. Systemic metabolic syndrome is present if a person has three or more of the five health problems listed: obesity, high blood pressure, high blood sugar, high serum triglycerides, and low serum high-density lipoprotein are associated with a diagnosis of

psoriasis.[105] Extracutaneous manifestation of psoriasis include nail abnormalities and psoriatic arthritis. Nail psoriasis is commonly misdiagnosed and treated as a fungal infection. Psoriatic arthritis affects the digital joints, especially the fingers and toes in an asymmetric pattern. The soft tissue of the digits may become swollen, producing a sausage-like digit that is usually acutely painful. This is referred to as dactylitis (see Fig. 13-10). Aside from nail changes and psoriatic arthritis, psoriasis can be associated with depression, alcoholism, obesity, insulin resistance, hypertension, myocardial infarction, Crohn's disease, obstructive pulmonary disease, nonalcoholic fatty liver disease, and coronary artery disease.[104,106–108]

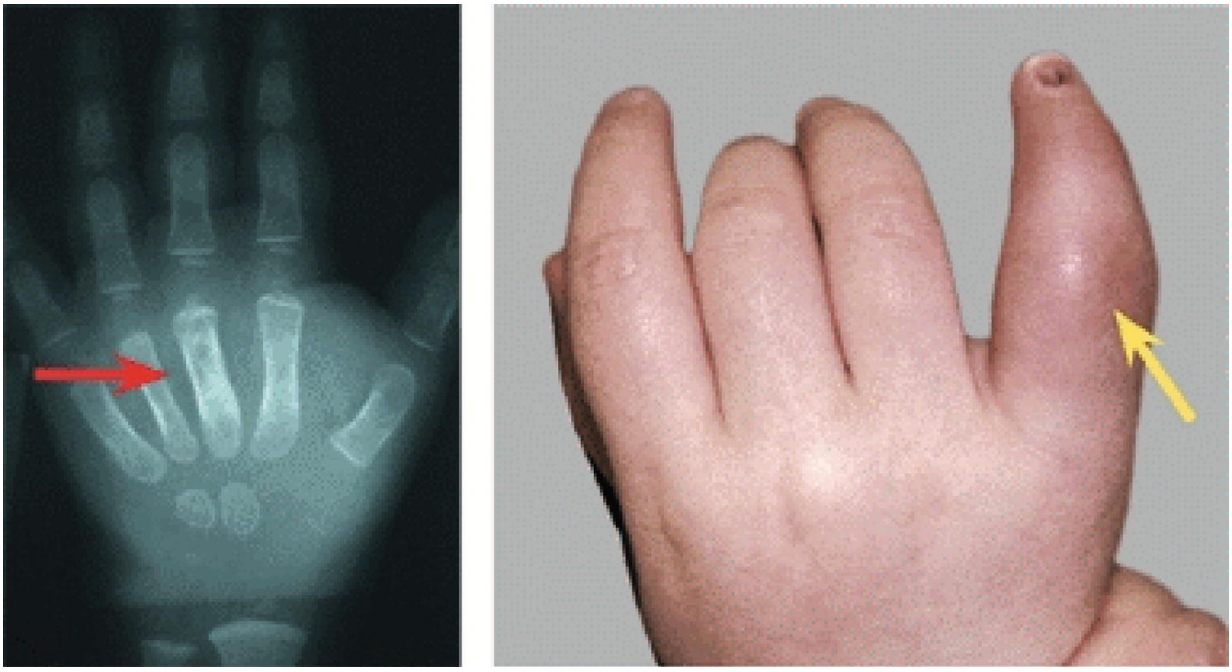


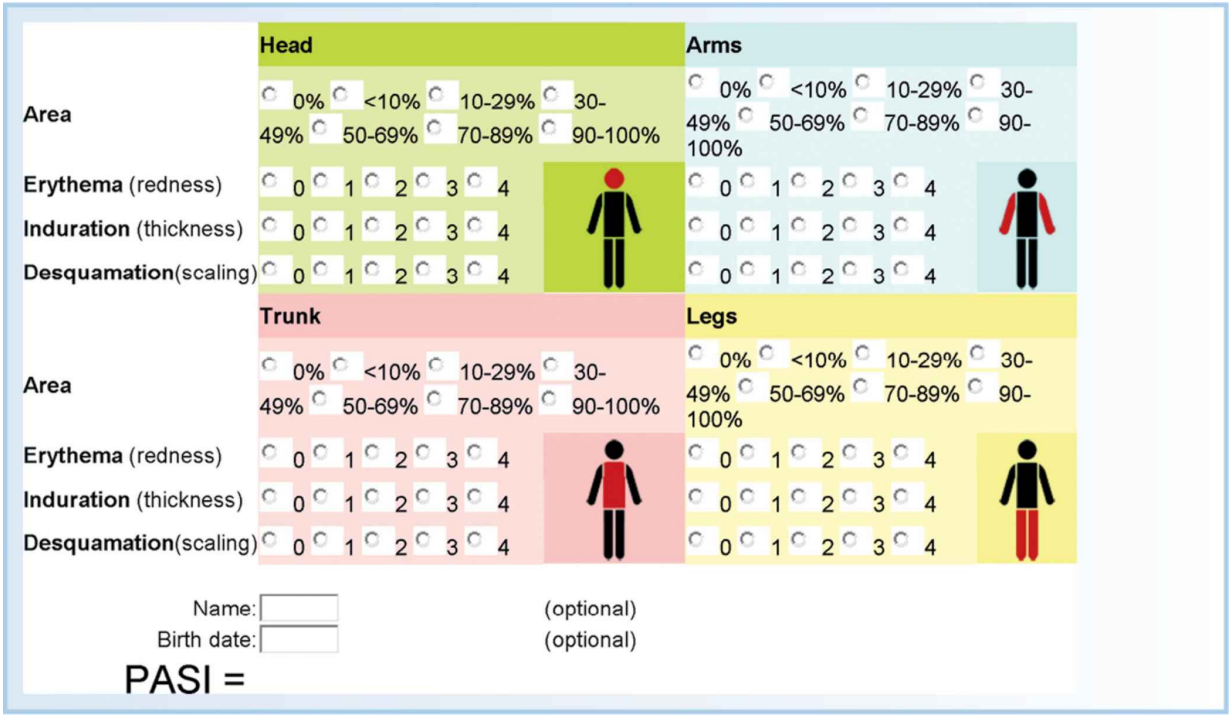
Figure 13-10. Dactylitis (as indicated by the *arrows*) is the term for when the soft tissue of a digit becomes swollen and the digit looks sausage-like. (Reprinted with permission from Lynn, T., Staheli, M.D. *Fundamentals of Pediatric Orthopedics*. Philadelphia, PA: Wolters Kluwer Health/Lippincott Williams & Wilkins, 2015.)

The wound care specialist should have a broad understanding of the forms of psoriatic lesions they may come across in all practice settings. Complex cases should be referred to a dermatologist for accurate assessment and appropriate interventions.

Chronic plaque psoriasis is the most common variant form of psoriasis. It is well-defined erythematous plaques with adherent silvery scale (see Fig. 13-11). The usual sites of involvement include the scalp, elbows, knees, lower lumbosacral area, and intergluteal cleft. However, other locations can be involved. The body surfaces areas involved can vary from minimal to extensive. The psoriasis area severity index (PASI) is an index used to express the severity of psoriasis. It combines the severity (erythema, induration, and desquamation) and percentage of affected area[100,104] (see Table 13-7).

Figure 13-11. Plaque psoriasis.

Table 13-7 Psoriasis Area Severity Index (PASI)



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Practice Point

Psoriasis plaques are composed of well-defined areas of dry, red, thickened skin covered with silvery-white scales.

Intertriginous Psoriasis

Intertriginous psoriasis can occur at the point of contact of any two skin folds; the inguinal fold, axilla, and external genitalia are the most common sites. It presents as well-demarcated shiny red plaques with minimal to no scale. Assess for signs of infection between the skin folds.

Pustular Psoriasis

Pustular psoriasis is an uncommon manifestation of psoriasis characterized by eruption of sterile pustules. It is often associated with intense pruritic and other skin manifestations, such as vesicles, purpura, and erythema multiform- like lesions predominantly on the palms or soles and often within or at the periphery of psoriatic plaques (Fig. 13-12). It may be localized or generalized. The disease severity and patient comorbidities should govern care and management. Other forms of psoriasis can occur and are beyond this chapter.

Figure 13-12. Pustular psoriasis. (Reprinted with permission from Council, M.L., Sheinbein, D., Cornelius, L.A. *The Washington Manual of*

Dermatology Diagnostics. Philadelphia, PA: Wolters Kluwer Health/Lippincott Williams & Wilkins, 2016.)

Periwound skin is a vulnerable area for psoriatic patients because of the Koebner's phenomenon, where traumatized skin more easily flares with psoriasis 1 or 2 weeks later. Maceration of the skin (due to overhydrating the stratum corneum keratin) by wound exudate and skin stripping due to adhesive dressings is also a source of skin damage and trauma. Liquid barrier films (film-forming liquid acrylates) can be used as a protective but flexible coating to cover tissue at risk of maceration.[104]

Multiple topical and systemic treatment options are available. Therapy is directed by the type of psoriasis one presents with and the severity. Mild to moderate forms of psoriasis can often be treated with topical steroids and steroid-sparing topical agents that may need to be supplemented with intralesional steroids or ultraviolet light. Systemic drugs and biologic agents are typically reserved for those with more severe presentations.[100,104]

Epidermolysis Bullosa

Epidermolysis bullosa is a group of fragile skin, mucosal, and internal organ–related genetic conditions that can be inherited as an autosomal dominant and recessive disorder that can be divided into four major subgroups (with over 20 variants) based on the level of blister formation[109–112]:

- Simplex or intraepidermal (epidermolytic)—superficial epidermal—mostly mild and often blisters in hot weather
- Junctional or lamina lucida level of the basement membrane with severe involvement of blisters on the hands, feet, and face at birth with some infants dying
- Dystrophic or sublamina densa of the basement where the disease is often severe with fusion of the fingers and toes, malnutrition and esophageal disorders, an early age death rate from infections, and cardiac complications but also a high incidence of squamous cell carcinomas (SCCs) if the affected individuals live into their 20s with these SCCs becoming more aggressive with age and subjects usually dying at a young age
- Kindler syndrome—a rare mixed variant

There is also an acquired skin condition called *epidermolysis bullosa acquisita* that resembles bullous pemphigoid, but these patients have milia associated with their blisters (Fig. 13-13).

Figure 13-13. Types of EB. (Reprinted with permission from Pope, E., Lara-Corrales, I., Mellerio, J.E., et al. *Epidermolysis Bullosa and Chronic Wounds: A Model for Wound Bed Preparation of*

Fragile Skin. Philadelphia, PA: Wolters Kluwer Health/Lippincott Williams & Wilkins, 2013.)

Practice Point

Check for cutaneous squamous cell carcinomas in persons with dystrophic junctional EB.

Treatment

Wound care treatment recommendations using the wound bed preparation paradigm are outlined in Table 13-8.

Table 13-8 Wound Care Recommendations for Persons with Epidermolysis Bullosa

Main Themes	Specific Themes	Specific Recommendations
	Select an appropriate dressing/ topical therapy based on the subtype of EB	Autolytic debridement: alginates, hydrogels Superficial critical colonization: silver, honey, polyhexamethylene biguanide Moisture balance with silicone coatings to prevent trauma, pain
	Evaluate the expected rate of healing or reassess wound goals of care	Reassess individuals not healing at the expected rate Low hemoglobin Low albumin Infection Systemic organ compromise
	Edge effect: If a wound is stalled or the edge/other areas appear atypical, consider a skin biopsy to rule out squamous cell carcinoma or other complications prior to considering active therapeutic options	Determine if wound is healable but stalled Consider advanced or active therapies Skin grafts Living skin equivalents (beware of potential HLA sensitization for future bone marrow transplant and other procedures) Biological agents
D. Provide organizational support	Consider a healthcare system support structure including specialized nurses, interprofessional clinics, and a structured approach to new cases	Each new case needs diagnosis and typing/subtyping ASAP Develop healthcare system support for new patients (existing models) Individual patients need community virtual interprofessional team http://www.internationalebforum.org

Used with permission from Pope, E., Lara-Corrales, I., Mellerio, J.E., et al. Epidermolysis Bullosa and Chronic Wounds: A Model for Wound Bed Preparation of Fragile Skin. *Advances in Skin & Wound Care* 26:177-88, 2013.

Practice Point

> The major issues to address in the care of a person with EB are pain, itch, and local skin and wound care.

The correction of the cause may include gene therapy in the future, but now bone marrow transplantation attempts have been disappointing due to the need for antirejection drugs that may increase the incidence of infection and susceptibility to malignancy as well as the fragile nature of the transplanted cells. Cofactors that may inhibit healing include the need for blood transfusions if the hemoglobin is low (especially under 80) and the need for nutritional support that may include a feeding tube at a very young age for supplementation.

Nutrition

For some persons with EB, the oral and esophageal mucosae are also affected. For newborns, sucking or eating can result in blisters while feeding (see Fig. 13-14) resulting in limited nutritional intake. Also, the pain from ulcerations on the lips and mouth can make it difficult for the person with EB to have adequate nutritional intake. Vitamin and mineral deficiencies may also be a problem. Having a dietician as part of the EB treatment team is important to help plan an appropriate diet and fluid intake. For persons with EB, while hemoglobin levels greater than 100 g/dL, for many individuals with severe EB and chronic wounds, would be ideal, a hemoglobin of 80 g/dL is a more realistic recommendation.[111]

Figure 13-14. EB of the oral cavity/mouth. (Reprinted with permission from Chung, E.K., Atkinson-McEvoy,

L.R., Lai, N.L., et al. *Visual Diagnosis and Treatment in Pediatrics*. Philadelphia, PA: Wolters Kluwer Health/Lippincott Williams & Wilkins, 2014.)

Patient-centered concerns often revolve around medication for itch (hydroxyzine syrup, diphenhydramine syrup) often given at night if they are sedating. Pain both nociceptive (consider acetaminophen prior to acetylsalicylic acid or narcotics, or neuropathic with gabapentin, pregabalin being useful for this pain and itch. All three sensations—itch and both nociceptive and neuropathic pain—may coexist in the same patient. Some of the newer antihistamines that are nondrowsy may be useful in these patients. Mobility is often an issue especially with mitten deformities that develop with fusion of the fingers and toes. Activities of everyday living often require assistance for many grooming or personal support workers for grooming and dressing.[109–112]

Skin and Wound Care

Best practice and consensus documents exist that form a basis for skin and local wound care of the EB patient.[109–111] Local wound care consists of treating these wounds as maintenance wounds with gentle local care avoiding adhesives other than silicone foams and tapes. Cleansing should be with compresses (ring gauze out after placing in water, saline or occasionally dilute acetic acid 0.5%, or chlorhexidine mouthwashes or mild PHMB solutions). Bathing can be used to remove adherent dressings with dilute bleach added for an antimicrobial effect.

New blisters should be broken with a sterilized needle, but the roof needs to be left on the surface of the drained blister. This is one exception where topical antibiotic creams including silver sulfadiazine can be used.[111] If any silver product is applied over large areas for prolonged periods of time, serum silver levels should be monitored, and these can reach very high levels if large areas of skin are treated over a prolonged period of time especially when these children are skinny and have a small stature and low weight. Conservative atraumatic debridement of slough is preferred to active surgical debridement that often results in pain.

Local infection should be diagnosed based on three or more NERDS criteria (see Chapter 7). Topical antimicrobial agents may include silver dressings, slow release iodine or antimicrobial gauze packing/dressings, and occasionally iodine-soaked ribbon gauze that should be fluffed and not stuffed. Foam dressings to absorb exudate should be nonadherent or have relatively atraumatic adhesives. If exudate is excessive, superabsorbent pads are often useful, but the presence of large amount of exudate may indicate deep and surrounding infection that can be identified by three or more STONEES criteria (see Chapter 7). Oral antibiotics are often tolerated best in liquid formulations with systemic intravenous antibiotics for resistant infections.

Moisture balance is often best with foam dressings. Dry wounds may benefit from hydrogels, but many wounds in these patients require absorptive dressings to avoid periwound maceration from the

excess local moisturizers. There is limited benefit from cellular skin replacement therapy, acellular matrices, or growth factors. Most surgeries heal best with rotational flaps or skin grafting with durable materials.

The holistic care of persons with EB requires compassion to advocate for healthcare system resources that are necessary for wound maintenance including rehabilitative assistance. There is also a need to link with EB support groups such as DebRA International (www.debra.org).

Practice Point

EB is a complex, multisystem disease without a cure that requires a coordinated interprofessional team approach to the whole person.

In the end analysis, give the patient the freedom to try dressings[113] that may or may not be optimal for wound healing. Patient comfort is paramount. If they want to use honey or other dressings, this may be an option. Measure the wounds to be treated with the experimental treatment, and monitor the wound characteristics every 2 to 4 weeks.

Biopsy of atypical areas of skin is the best way to diagnose squamous cells early. The presence of a squamous cell that is aggressive may require limb amputation to save a life particularly in the recessive dystrophic EB patients where the defect in type 7 collagen in the anchoring filaments leads to increased areas of blistering as the patient's general health deteriorates. Be a facilitator of care not a dictator of treatment. Foster adherence with patient-centered care that supports patient empowerment and self-management.

Skin Failure, Terminal Skin Ulcers, and Skin Changes at Life's End

The concepts of skin failure, terminal skin ulcers, and skin changes at life's end (SCALE) remain controversial.[114] There are healthcare professionals who do not believe that these exist and others who are equally passionate that these are real clinical phenomenon. Lack of consensus and clarity regarding whether or not these concepts exist or what they are continues to exist in the literature. The literature supporting skin failure[115–128] and terminal ulcers[129–132] is very limited with much being expert opinion and a paucity of empirical evidence. There is some data about terminal ulcers[129–132] as well as the Delphi consensus process used to develop the SCALE document.[133–136] A summary of the current status of these concepts has been published[114] and can be found in Table 13-9.

Table 13-9 The Literature at a Glance: A Summary of Terminal Ulcers, Scale, and Skin Failure Concepts

Term (Year Published)	Definition	Characteristics/ Comments	Evidence/References
KTU (1989)	The KTU is shaped like a pear; predominately on the coccyx or sacrum. Red, yellow, and black. Onset of a KTU is sudden.[129]	Life expectancy from 2 weeks to several months.[129] Other locations include hip, heel, ischium. Additional shapes: butterfly, horseshoe. Borders are irregular. Presenting characteristics are a larger area with superficial depth.	One retrospective case review in long-term care from 1983 to 1988 (51 patients who died) after clinical observation of skin changes.[129]
Skin failure	An event in which the skin and underlying tissue die; attributable to hypoperfusion that occurs concurrent with severe dysfunction or failure of other organ systems.[121]	Can be categorized as chronic, end stage, or acute.[121] Life expectancy can vary. Generally over a bony prominence but can occur anywhere on the body. This condition is unavoidable in most instances. It is unclear whether this condition is independent of KTU, TB-TTI, or DTI.	One retrospective study of outcomes (180-day mortality) in 74 individuals. Brown[137] found PI development rates of 66.7% in intensive care, 75% in acute, and 66.7% in long-term care. No deaths were directly attributable to presence of a PI.[137]

Term (Year Published)	Definition	Characteristics/ Comments	Evidence/References
ASF (2006)	Describes the hypoperfusion state that leads to tissue death that occurs simultaneously to a critical illness.[122]	Statistically significant and independent predictors for ASF in ICU patients from the logistic regression model were pulmonary artery disease, mechanical ventilation for more than 72 hours, respiratory failure, liver failure, and severe sepsis/ septic shock.[122]	One retrospective case–control study[122] in two magnet medical centers in northeast US ($N = 450$). Mean length of stay was 9 days; mean Braden Scale score on admission to ICU was 14. Of all 450 patients, 150 had PIs; of those, 82 patients (54.7%) had sacral PI. Half of the PIs ($n = 75$, 50%) were staged as DTI. Stage 2 ($n = 43$, 29%) was the next most common. The majority of PIs ($n = 101$, 67%) developed in the first week of ICU admission.[122]
SCALE (2008)	A mnemonic used to describe a group of clinical phenomena.[135] "Physiological changes that occur as a result of the dying process may affect the skin and soft tissues and may manifest as observable (objective) changes in skin color, turgor, or integrity, or as subjective symptoms such as localized pain."[135]	The changes can be unavoidable and may occur with the application of appropriate interventions that meet or exceed the standard of care.[135] Recommended: research to identify the mechanisms for the proposed decreased hypoperfusion and oxygenation of the skin and soft tissues.[135] Further, distinguish skin and soft tissue damage associated with SCALE from other skin disorders not associated with skin organ compromise or the end of life.[135]	Modified 3-phase Delphi process used to develop 10 consensus statements originally drafted by 18 key opinion leaders, which were then reviewed by 49 international reviewers (2008),[133] and 52 international reviewers reached consensus on the 10 final statements (2010).[135] Review article with a case review of a long-term care resident.[136]
TB-TTI (2010)	Spontaneously appearing skin alterations (rapid evolution, speed of enlargement and progression, appearance in areas of little to no pressure such as shins and thighs, and mirror imaging found in patients at the end of life). Unavoidable occurrence—one that is related to organ failure at the end of life.[132]	Bruise-like appearance (pink, purple, or maroon color); may be butterfly pattern. Can be linear striations on legs that extend downward or on thoracic or lumbar spine that present horizontally. May or may not be over bony prominence. Skin remains intact and does not break down. Can be confused with a DTPI.	Retrospective chart reviews from a 10-bed palliative care unit after clinical recognition of the skin changes (2010; $N = 22$).[131] Larger retrospective study (2012; $N = 80$).[132]

ASF, acute skin failure; DTI, deep tissue injury; DTPI, deep tissue pressure injury; ICU, intensive care unit; KTU, Kennedy terminal ulcer; PI, pressure injury; SCALE,

Skin Changes At Life's End; TB-TTI, Trombley-Brennan terminal tissue injury.
Used with permission Ayello, E.A., Levine, J.M., Langemo, D., et al. Reexamining the Literature on Terminal Ulcers, SCALE, Skin Failure, and Unavoidable Pressure Injuries. *Advances in Skin and Wound Care* 32(3):109-21, March 2019. © The authors.

Skin Failure

Healthcare providers have raised the question as to whether or not the skin can fail. Early discussion of skin failure as a concept was in 1989 by Goode and Allman who noted “multiple/multiorgan failure as a terminal stage of many diseases that occurs as the body wastes away.”[115] They concluded that the skin is susceptible to failure, and death must be considered in multiorgan death syndrome.[115] The term skin failure was again used in 1991 by LaPuma.[116] Patients who are at end stages of life and in the intensive care setting were the focus of an editorial in 1993 regarding skin and underlying tissue damage.[117] Leijten et al. discussed chronic skin failure in 1996 related to older adults with multiple comorbidities that can lead to pressure injuries, especially at the end of life.[119] Hobbs et al. discussed skin failure at a national program as an abstract in 2000.[120] Witkowski and Parish,[118] also in 2000, asked, “If the heart, lungs, and kidneys are failing, is it not logical that the body’s cover would also show signs of failure?”

The definition of skin failure has not been well defined clinically or universally accepted in the healthcare setting. Along with presentation at national conferences in the early 2000s, the first written definition of Langemo’s definition of skin failure was in her 2006 article.[121] Skin failure was defined as “an event in which the skin and underlying tissue die due to hypoperfusion that occur concurrent with severe dysfunction or failure of other organ systems.”[121] The definition was further described as three types of events that trigger skin failure: acute skin failure (ASF), chronic skin failure, and end-stage skin failure.[121] Diagnostic testing is currently not available to detect tissue necrosis in its decisive stages.

- ASF is an event in which skin and underlying tissue die due to hypoperfusion concurrent with a critical illness.
- Chronic skin failure is when skin and underlying tissue die due to hypoperfusion concurrent with an ongoing chronic disease state.
- End-stage skin failure is when skin and underlying tissue die due to hypoperfusion concurrent with the end of life.[121]

Several healthcare providers raise the question as to whether or not the skin can fail.[123–127] Empirical evidence about ASF has been published by Delmore and colleagues.[122] Their retrospective case-controlled study of 552 ICU patient in the United States shows independent predictors of ASF as being peripheral arterial disease, mechanical ventilation for greater than 72 hours, respiratory or liver failure, and several sepsis/septic shock. They cautioned that due to limited evidence, clinicians might be misidentifying ASF as pressure injuries. The need for dermatology and skin/wound care professionals to work together to come to consensus on a definition and diagnostic criteria of skin failure can be found in the literature.[114,122–124]

Terminal ulcers

While some healthcare professionals do not believe in the concept of terminal ulcers,[138] the reality is that others do[139] including in the United States; the Centers for Medicare and Medicaid Services (CMS) does recognize them as a clinical phenomenon.[140] The first of these ulcers was reported in the literature in 1989[129] based on retrospective case review of 51 residents who died with these “terminal lesions.” Although the mechanisms for these “butterfly”-shaped ulcers found mostly in the sacral area was not clearly delineated nor understood, these skin conditions became known as Kennedy terminal ulcers (KTU). Further empirical evidence about KTUs is yet to be published.

Data on skin alternations seen in palliative care inpatients has been reported by Trombley and Brennan.[131,132] The first study of 22 patients provided clinical descriptions of what they called “butterfly”-shaped pink, purple, or maroon skin tissue injuries in the pelvic area as well as linear striations on the legs and horizontal striations in the lumbar or thoracic spine regions. The authors believe that terminal ulcers developed despite prevention measures and were not pressure injuries. A further study with a larger sample of 80 patients led the researchers to affirm that these terminal ulcers[131,132] were not pressure injuries and that these clinically observed skin changes were seen in patients within days of death (Fig. 13-15).

A

Figure 13-15. Skin failure. (**A:** Used with permission from S.Baranoski and **B:** Used with permission from M. Brennan.)

Skin Changes at Life's End

The literature on skin changes at life's end is limited. In 2008, key opinion leaders convened[134] and stated that "general agreement was reached that like any other organ of the body, skin is subject to a loss of integrity due to internal and external insults. Contrary to popular myth, not all pressure ulcers are avoidable." Because the panel concluded that the current appraisal of the complex skin changes at life's end and terminal pressure injuries (including the KTU) was limited, they undertook an international consensus modified Delphi process to validate 10 statements about the clinical observation of skin changes in persons at life's end that were not pressure injuries or skin failure nor as a result of lack of care. Although one retrospective Swedish study in 2017[141] reported predictors of developing pressure injuries in patients at end of life, no additional data has been published since the original SCALE documents.

Summary

The skin is the largest organ in the body and one of the most complicated. It has many roles in the maintenance of life and health but also has many potential problems and many possible skin disorders. The skin is exposed daily to environmental irritants and chemicals as well as physical and mechanical injury, any of which may lead to impaired skin integrity. This chapter provided an overview of some common skin conditions: dermatitis, MASD, skin tears, psoriasis, epidermolysis bullosa, and various forms of skin failure and/or terminal ulcers. Identifying the correct cause and developing an appropriate evidence-based plan of care are critical for patient care outcomes.

PATIENT SCENARIO

Clinical Data

The patient is a 74-year-old hospitalized female for COPD who has urinary incontinence. Due to her difficulty with breathing, she does not have much energy and is very reluctant to move and to get out of bed to use the commode. Getting on the bedpan is also difficult for her.

The staff noticed skin discoloration (erythema) from contact with urine. An IAD skin protocol was implemented that consisted of gentle skin cleansing with a one-step skin cleansing cloth (Fig. 13-16). This was followed with application of zinc oxide as needed after incontinent episodes.

Figure 13-16. Day 0.

Because the staff was concerned about her skin integrity, they started her on a pressure injury prevention protocol. An appropriate support surface was ordered for pressure relief. The staff developed a turning and repositioning schedule within her comfort level, taking into consideration her breathing difficulties. The patient was encouraged to move, reposition herself, and get up and out of bed with assistance. Six days later, skin improved (Fig. 13-17).

Figure 13-17. Note the improvement of her skin 6 days later.

PATIENT SCENARIO

Clinical Data

Mr. JP is a 75-year-old white male with a history of cardiac disease, COPD, and renal disease. He has a pacemaker and a defibrillator for the past 5 years. He has a 20-year history of being on warfarin. He is frequently in and out of the hospital with pulmonary and respiratory distress. Due to his cardiac problems, he has frequent bouts of fluid retention causing him to have edematous legs and feet. He was at home ambulating to the bathroom when he knocked his walker over onto his edematous foot. Although he did not fall, 24 hours later he complained of pain, discoloration, and more swelling to his left foot (Fig. 13-18). The right foot was normal. His wife brought him to the emergency room where he was subsequently admitted to a medical surgical unit.

Figure 13-18. Initial presentation of left foot.

There, he was evaluated by a surgeon who said “this needs to come off” and recommended that the foot be immediately amputated. Mr. JP and his wife refused. JP’s primary physician then took over the case, and after discussion with the family, a wound care doctor and podiatrist were called in to handle the case. The joint consultation of these physicians resulted in a recommendation of a different approach. The treatment plan that they presented was as follows: keeping the foot elevated, drying out the blister on the top of the foot by using povidone–iodine, and allowing the blood in the foot to dissipate on its own. Basically, they felt that the best treatment was a slow medical management, with good skin and wound care, and getting him up and mobile over time was the best approach. So this conservative management plan was implemented (Fig. 13-19).

Figure 13-19. Left foot after 2 months of treatment.

During his initial skin assessment on the medical surgical unit, the nurse noted numerous areas of skin tears and bruising on his entire back (Fig. 13-20). Mr. JP said that this occurred when he was dragged from the ambulance stretcher to the emergency department gurney. At that time, Mr. JP immediately complained “my back, my back.” The ED staff did nothing. The medical–surgical unit nurse also noted that on his left arm above the elbow was a large full-thickness skin tear. Mr. JP had no idea how that skin tear occurred (Fig. 13-21). As you recall, the upper extremities are the most common place for skin tears. Because the skin tear is full thickness, it was documented as a type 3 using the ISTAP classification system. The numerous skin tears on his back were classified as types 2 and 3.

Figure 13-20. Initial presentation of skin tears on his back.

Figure 13-21. A initial presentation of skin tear on left arms.

Case Discussion

Mr. JP had numerous skin care problems that needed to be addressed. Because he already has several skin tears, Mr. JP is assessed as being at high risk for further developing more skin tears. Assessment of his back revealed seven skin tears: four type 3 and three type 2. His left arm also revealed a type 3 skin tear (ISTAP classification system). Implementing strategies to prevent more skin tears from occurring included protecting other areas of risk on his arms by encouraging him to wear arm protectors to add a layer of padding to protect his skin (Fig. 13-22). This also has the added benefit of keeping him warm as he often complained of feeling chilly. As part of the prevention strategies, staff were reminded of proper

transfer techniques such as not grabbing Mr. JP's arms or dragging his back across the mattress when assisting him out of bed.

Figure 13-22. Padding the arms for protection and exudate strikethrough on dressings. Dressing needs to be changed.

Local care of Mr. JP's skin tears was required. After cleansing with normal saline, a soft nonadherent silicone dressing was applied to each of the skin tears on his back and arm. Nonadherent silicone dressings were chosen as they would not cause any further skin damage during dressing removal. Moisturizing creams were used over other areas of senile purpura and areas of xerosis (dry skin) that were thought to be at risk. Mr. JP complained about the leaking and wetness from the dressings on his arm. The dressing was overwhelmed with exudate (see Fig. 13-22). Staff were reminded to change the dressings every 3 days or more frequently if needed in

order to address the issue of dressing strikethrough (Fig. 13-23). After 2 weeks of using the nonadherent dressings, changing them when needed, before fluid was leaking, and using appropriate transfer techniques, many of the skin tears on his back healed (Fig. 13-24).

Figure 13-23 Better padding of the arm with no dressing strikethrough on dressing.

Figure 13-24. Skin tears on his back after 2 weeks of treatment.

Mr. JP remained in the hospital for 2 weeks and was then transferred to a rehabilitation facility. During that time, he developed no new skin tears, and a wound care nurse came to see him frequently and continued to treat the skin tears with use of nonadherent silicone dressings. Figure 13-25 shows the healed skin tears on his left arm after 6 weeks. After 2 months of the conservative treatment for his left foot, most of the discoloration and edema and the blood blister is resolved. Most importantly, Mr. JP has been discharged home and still has his foot and leg.

Figure 13-25. Left arm skin tear after 6 weeks of treatment with nonadherent dressing. The original skin tear has healed. Note scarring from previous skin tears.

Show What You Know

1. **Which one of the following patients is most at risk for skin tear injury?**
   A. 22-year-old male postoperative for an inguinal hernia repair
   B. 37-year-old male with a fractured humerus
   C. 64-year-old female 3 days postcataract extraction
   D. 72-year-old female with rheumatoid arthritis on steroid therapy

2. **Using the ISTAP system, a partial-thickness flap loss that cannot be repositioned to cover the wound bed is classified as:**
   A. type 1.
   B. type 2.
   C. type 3.
   D. type 4.

3. **Which of the following interventions for a resident in a long-term care facility with a skin tear on the lower right leg should you question?**
   A. Clean the patient daily using detergent.
   B. Pad the wheelchair arm and leg supports.
   C. Apply a nonadherent dressing to the skin tear.
   D. Encourage the patient to wear soft, fleece-lined pants.

4. **A patient has skin damage from perspiration and friction under his abdominal pannus and the culture comes back as *Candida*. The best way to classify this skin damage is as:**
   A. peristomal skin damage.
   B. periwound maceration.
   C. incontinence-associated dermatitis.
   D. intertriginous dermatitis.

5. **The most common variant form of psoriasis is:**

A. pustular psoriasis.
B. chronic plaque psoriasis.
C. nail psoriasis.
D. intertriginous psoriasis.

6. **is defined as an event in which the skin and underlying tissue die due to hypoperfusion that occur concurrent with severe dysfunction or failure of other organ systems.**
   A. Xerosis
   B. Skin failure
   C. Skin tear
   D. Pruritus

7. **Target hemoglobin level for a person with EB should be:**
   A. 50 g/dL.
   B. 60 g/dL.
   C. 70 g/dL.
   D. 80 g/dL.

8. **Cutaneous squamous cell carcinoma is a serious problem in persons with what types of EB.**
   A. Simplex
   B. Dystrophic
   C. Kindelar
   D. Koebner

Answers to these questions are found on page 684.

References

1. Alavi, A., Sibbald, R.G., Ladizinski, B., et al. Wound-Related Allergic/Irritant Contact Dermatitis. Advances in Skin & Wound Care 29(6):278-86, 2016.
2. www.aad.org/practicecenter/quality/clinical-guidelines/atopic-dermatitis. Accessed March 5, 2020.
3. Woo, K.Y., Beeckman, D., Chakravarthy, D. Management of Moisture-Associated Skin Damage: A Scoping Review. Advances in Skin & Wound Care 30(11):494-501, 2017.
4. Beele, H., Smet, S., Van Damme, N.,et al. Incontinence-associated Dermatitis: Pathogenesis, Contributing Factors, Prevention and Management Options. Drugs & Aging 35(1):1-10, 2018.
5. Wong, V.K., Stotts, N., Hopf, H.W., et al. “Changes in Heel Skin Temperature Under Pressure in Hip Surgery Patients.” Advances in Skin & Wound Care 24(12): 562-70, 2011.
6. Gray, M., Black, J., Baharestani, M., et al. “Moisture Associated Skin Damage, Overview & Pathophysiology.” Journal of Wound, Ostomy, and Continence Nursing 38(3):233-41, 2011.
7. Black, J., Gray, M., Bliss, D. “Part 2: Incontinence Associated Dermatitis and Intertriginous Dermatitis.” Journal of Wound, Ostomy, and Continence Nursing 38(4):359-70, 2011.
8. Gray, M., Bohacek, L., Weir, D., et al. “Moisture vs Pressure. Making Sense Out of Perineal Wounds.” Journal of Wound, Ostomy, and Continence Nursing 34(2):134-42, 2007.
9. Gray, M., Bliss, D.Z., Doughty, D.B., et al. “Incontinence-Associated Dermatitis—A Consensus.” Journal of Wound, Ostomy, and Continence Nursing 34(1):45-54, 2007.
10. Defloor, T., Schoonhoven, L., Fletcher, J., et al. “Statement of the European Pressure Ulcer Advisory Panel—Pressure Ulcer Classification. Differentiation Between Pressure Ulcers and Moisture Lesions.” Journal of Wound, Ostomy, and Continence Nursing 32(5): 302-6, 2005.
11. Zulkowski, K. “Perineal Dermatitis versus Pressure Ulcer: Distinguishing Characteristics.” Advances in Skin & Wound Care 21(8):382-8; quiz 389-90, 2008.
12. Mahoney, M., Rozenboom, B., Doughty, D. “Challenges in Classification of Gluteal Cleft and Buttock Wounds.” Journal of Wound, Ostomy, and Continence Nursing 40(3):239-45, 2013.
13. Colwell, J., Ratcliff, C., Goldberg, M., et al. “MASD Part 3: Peristomal Moisture-Associated Dermatitis and Periwound Moisture-Associated Dermatitis: A Consensus.” Journal of Wound, Ostomy, and Continence Nursing 35(5):541-53, 2011.
14. McNichol, L.L., Ayello, E.A., Phearman, L.A., et al. Incontinence Associated Dermatitis: State of the Science and Knowledge Translation. Advances in

Skin & Wound Care 31(11):502-13, 2018.
15. Beeckman, D., Bliss, D., Buckley, B., et al. Management Using Continence Products. In P. Abrams, L. Cardozo, L. Wagg, et al., eds. Incontinence ICI-ICS, 6th ed. Bristol, UK: International Continence Society, 2017.
16. Beeckman, D., Van Lancker, A., Van Hecke, A., et al. A Systematic Review and Metaanalysis of Incontinence-Associated Dermatitis, Incontinence, and Moisture as Risk Factors for Pressure Ulcer Develop. Research in Nursing and Health 37(3):204-18, 2014. doi: 10.1002/nur.21593.
17. Demarre, L., Verhaeghe, S., Van Hecke, A., et al. Factors Predicting the Development of Pressure Ulcers in an At-Risk Population Who Receive Standardized Preventive Care: Secondary Analyses of a Multicentre Randomised Controlled Trial. Journal of Advanced Nursing 71(2), 391-403, 2015. doi: 10.1111/jan.12497.
18. Gray, M., Giulano, K. Incontinence-associated Dermatitis, Characteristics and Relationship to Pressure Injury. A Multisite Epidemiologic Analysis. Journal of Wound, Ostomy, and Continence Nursing 45(1):63-7, 2018.
19. Larner, J., Matar, H., Goldman, V.S., et al. Development of a Cumulative Irritation Model for Incontinence-Associated Dermatitis. Archives of Dermatological Research 307:39-48, 2015.
20. Bender, J.K., Faergemann, J., Skold, M. Skin Health Connected to the Use of Absorbent Hygiene Products: A Review. Dermatology and Therapy 7(3):319-30, 2017.
21. Campbell, J.L., Coyer, F.M., Osborn, S.R. Incontinence-associated Dermatitis: A Cross Sectional Prevalence Study in the Australian Acute Care Hospital Setting. International Wound Journal 13(3):403-11, 2016.
22. Ayello, E.A., Sibbald, R.G., Quiambao, P.C.H., et al. Introducing a Moisture-Associated Skin Assessment Photo Guide for Brown Pigmented Skin. World Council of Enterostomal Therapists Journal 34(2):18-25, 2014.
23. Nix, D.H. Validity and Reliability of the Perineal Assessment Tool. Ostomy/Wound Management 48(2):43-6,48-9, 2002.
24. Borchert, K., Bliss, D.Z., Savik, K., et al. The Incontinence-Associated Dermatitis and its Severity Instrument: Development and Validation. Journal of Wound, Ostomy, and Continence Nursing 37(5):527-35, 2010.
25. Brown, D.S. Perineal Dermatitis Can We Measure It? Ostomy/Wound Management 39(7):28-30, 1993.
26. Kennedy, K.L., Lutz, L. Comparison of the Efficacy and Cost Effectiveness of three Skin Protectants in the Management of Incontinence Dermatitis. In: Proceeding of the European Conference on Advances in Wound Management; October 4, 1995; Amsterdam.
27. Clarke-O'Neill, S., Fabrot, A., Lagerstedt Eidrup, M.L., et al. An Exploratory Study of Skin Problems Experienced by UK Nursing Home Residents Using Different Pad Designs. Journal of Wound, Ostomy, and Continence Nursing 42(4):379-88, 2015.

28. Bliss, D.Z., Hurlow, J., Defalu, J., et al. Refinement of an Instrument for Assessing Incontinent-Associated Dermatitis and its Severity for Use with Darker-Toned Skin. Journal of Wound, Ostomy, and Continence Nursing 41(4):365-70, 2014.
29. Beeckman, D. Global IAD Expert Panel. Incontinence-Associated Dermatitis: Moving Prevention Forward. Wounds International. 2015. Available at: www.woundsinternational.com/consensus-documents/view/incontinence-associated-dermatitis-moving-prevention forward
30. Beeckman, D., Van den Bussche, K., Alves, P., et al. Towards an International Language for Incontinence-Associated Dermatitis (IAD): Design and Evaluation of Psychometric Properties of the Ghent Global IAD Categorization Tool (GLOBIAD) in 30 Countries. British Journal of Dermatology 178(6):1331-40, 2018.
31. Beeckman, D. A Decade of Research on Incontinence-Associated Dermatitis (IAD): Evidence, Knowledge Gaps and Next Steps. Journal of Tissue Viability 26:47-56, 2017.
32. Gray, M. Incontinence-Related Skin Damage: Essential Knowledge. Ostomy/Wound Management 53(12):28-32, 2007.
33. Kottner, J., Beeckman, D. Incontinence-Associated Dermatitis and Pressure Ulcers in Geriatric Patients. Giornale Italiano di Dermatologia e Venereologia 150(6):717-29, 2015.
34. Doughty, D.B. Promoting Continence: Simple Strategies with Major Impact. Ostomy/Wound Management 49(12):46-52, 2003.
35. Gray, M., Kent, D., Ermer-Seltun, J., et al. Assessment, Selection, Use, and Evaluation of Body-Worn Absorbent Products for Adults with Incontinence: A WOCN Society Consensus Conference. Journal of Wound, Ostomy, and Continence Nursing 45(3):243-64, 2018.
36. Brunner, M., Droegemueller, C., Rivers, S., et al. Prevention of Incontinence-Related Skin Breakdown for Acute and Critical Care Patients: Comparison of Two Products. Urologic Nursing 32(4):214-9, 2012.
37. Zhai, H., Maibach, H.I. Anti-Irritants Agents for the Treatment of Irritant Contact Dermatitis: Clinical and Patent Perspective. Recent Patents on Inflammation & Allergy Drug Discovery 6(3):169-85, 2012.
38. Foureur, N., Vanzo, B., Meaume, S., et al. Prospective Aetiological Study of Diaper Dermatitis in the Elderly. The British Journal of Dermatology 155(5):941-6, 2006.
39. Shaked, E., Gefen, A. Modeling the Effects of Moisture-Related Skin-Support Friction on the Risk for Superficial Pressure Ulcers During Patient Repositioning in Bed. Frontiers in Bioengineering and Biotechnology 1:9, 2013. Retrieved from https://www.frontiersin.org/articles/10.3389/fbioe.2013.00009/full
40. Scardillo, J., Aronovitch, S.A. Successfully Managing Incontinence-Related Irritant Dermatitis Across the Lifespan. Ostomy/Wound Management 45(4):36-44, 1999.

41. Noonan, C., Quigley, S., Curley, M.A.Q. Skin Integrity in Hospitalized Infants and Children a Prevalence Study. Journal of Pediatric Nursing 21(6):445-53, 2006.
42. Lewis-Byers, K., Thayer, D. An Evaluation of Two Incontinence Skin Care Protocols in a Long-Term Care Setting. Ostomy/Wound Management 48(12):44-51, 2002.
43. Bliss, D.Z., Bland, P., Wiltzin, K., et al. Incontinence Briefs Containing Spiral-Shaped Fiber Acidify Skin pH of Older Nursing Home Residents at Risk for Incontinence-Associated Dermatitis. Journal of Wound, Ostomy, and Continence Nursing 44(5):475-80, 2017.
44. Sibbald, R.G., Kelley, J., Kennedy-Evans, L.K., et al. A Practical Approach to the Prevention and Management of Intertrigo, or Moisture-Associate Skin Damage due to Perspiration: Expert Consensus on Best Practice. Wounds Care Canada 11(2), 2013.
45. Janniger, C.K., Schwartz, R.A., Szepietowski, J.C., et al. “Intertrigo and Common Secondary Skin Infections.” American Family Physician 72:833-38, 2005.
46. LeBlanc, K., Campbell, K., Beeckman, D., et al. Best Practice Recommendations for the prevention and Management of Skin Tears in Aged Skin. Wounds International 45:540-2, 2018. Available at: www.woundsinternational.com
47. LeBlanc, K., Baranoski, S., Christensen, D., et al. “State of the Science: Consensus Statements for the Prevention, Prediction, Assessment, and Treatment of Skin Tears.” Advances in Skin & Wound Care 24(9):2, 2011.
48. LeBlanc, K., Baranoski, S., Langemo, D., et al. “A Descriptive Cross Sectional International Study to Explore Current Practices in the Assessment, Prevention and Treatment of Skin Tears.” International Wound Journal 11(4):424-30, 2004.
49. Woo, K.Y. “Trends in Wound Management.” Advances in Skin & Wound Care 26(12):538, 2013.
50. Garcia, E. Skin Tears: Assessment and Management. Adelaide, Australia: Joanna Briggs Institute, 2013.
51. Krasner, D.L., Rodeheaver, G.T., Sibbald, R.G., et al. “International Interprofessional Wound Caring.” in D.L. Krasner, G.T. Rodeheaver, R.G. Sibbald, et al., eds. Chronic Wound Care: A Clinical Source Book for Healthcare Professionals, Vol. 3, 5th ed. Malvern, PA: HMP Communications, 2012.
52. LeBlanc, K., Baranoski, B., Christensen, D., et al. “International Skin Tear Advisory Panel: Putting It All Together, a Tool Kit to Aid in the Prevention. Assessment and Treatment of Skin Tears.” Advances in Skin & Wound Care 26(10):451, 2013.
53. LeBlanc, K., Christensen, D., Orstead, H., et al. “Best Practice Recommendations for the Prevention and Treatment of Skin Tears.” Wound Care Canada 6(1):14, 2008.

54. LeBlanc, K., Baranoski, B., Holloway, S., et al. “Validation of a New Classification System for Skin Tears.” Advances in Skin & Wound Care 26(6):264, 2013.
55. da Silva, C.V.B., Campanili, T.C.G.F., LeBlanc, K., et al. Cultural Adaptation and Content Validity of ISTAP Skin Tear Classification for Portuguese in Brazil. Revista Estima 16:1-7, 2018.
56. Sun, Q., He, Y., Liu, K., et al. Recent Advances in Terahertz Technology for Biomedical Applications. Quantitative Imaging in Medicine and Surgery **7**:345, 2017.
140. Chai, J., Ge, J., Zou, J. Effect of Autologous Platelet-Rich Plasma Gel on Skin Flap Survival. Medical Science Monitor 25:1611-20, 2019.
57. Van Tiggelen, H., LeBlanc, K., Campbell, K., et al. Standardising the Classification of Skin Tears: Validity and Reliability Testing of the International Skin Tear Advisory Panel (ISTAP) Classification System in 44 Countries. British Journal of Dermatology, 2019. doi: 10.1111/bjd.18604.
58. Carville, K., Lewin, G., Newall, N., et al. “STAR: A Consensus for Skin Tear Classification.” Primary Intention: The Australian Journal of Wound Management 15(1):18, 2007.
59. Carville, K., Smith, J. “Report on the Effectiveness of Comprehensive Wound Assessment and Documentation in the Community.” Primary Intention: The Australian Journal of Wound Management 12:41, 2004.
60. Holmes, R.F., Davidson, M.W., Thompson, B.J., et al. “Skin Tears: Care and Management of the Older Adult at Home.” Home Healthcare Nurse 31(2): 90-103, 2013.
61. Hsu, M., Chang, S. “A Study on Skin Tear Prevalence and Related Risk Factors Among Inpatients [Chinese].” Tzu Chi Nursing Journal 9(4):84-95, 2010.
62. Koyano, K., Nakagami, G., Iizaka, S., et al. Exploring the prevalence of skin tears and skin properties related to skin tears in elderly patients at a long-term medical facility in Japan. International Wound Journal 13:189-97, 2016. doi: 10.1111/iwj.12251.
63. LeBlanc, K., Christensen, D., Cook, J., et al. Prevalence of Skin Tears in a Long-Term Care Facility. Journal of Wound Ostomy & Continence Nursing 40(6):580-4, 2013.
64. Skiveren, J., Wahlers, B., Bermark, S. Prevalence of Skin Tears in the Extremities Among Residents in a Nursing Home in Denmark. Journal of Wound Care 26(2):S32-6, 2017.
65. Woo, K., Sear, K., Almost, J., et al. Exploration of Pressure Ulcer and Related Skin Problems Across the Spectrum of Health Settings in Ontario using Administrative Data. International Wound Journal (1):24-30, 2015.
66. Woodbury, M.G., Houghton, P.E. “Skin Tears in Long-Term Care: Effectiveness of Skin Care Protocols on Prevalence.” Ostomy Wound Management 50(10):22-4, 26, 28, 2004.

67. Hanson, D., Hunter, S., Anderson, J., et al. “Skin Tears in Long-Term Care: Effectiveness of Skin Care Protocols on Prevalence.” Advances in Skin & Wound Care 18(2):74, 2005.
68. White, W. Skin Tears: A Descriptive Study of the Opinions, Clinical Practice and Knowledge Base of RNs Caring for the Aged in High Care Residential Facilities. Primary Intention: The Australian Journal of Wound Management 9(4):138-49, 2001.
69. White, M.W., Karam, S., Cowell, B. “Skin Tears in Frail Elders: A Practical Approach to Prevention.” Geriatric Nursing 15(2):95-9, 1994.
70. Ratliff, C.R., Fletcher, K.R. “Skin Tears: A Review of the Evidence to Support Prevention and Treatment.” Ostomy Wound Management 53(3):32-4, 36, 38-40, 2007.
71. Payne, R.L., Martin, M. “Skin Tears: The Epidemiology and Management of Skin Tears in Older Adults.” Ostomy Wound Management 26:26-37, 1990.
72. McGough-Csarny, J., Kopac, C. A. “Objectives I: Risk Factor Identification. Skin Tears in Institutionalized Elderly: An Epidemiological Study… Including Commentary by Van rijswijk L, Kirsner R, Bolton L, McNees P with author response.” Ostomy Wound Management 44(3):14S, 1998.
73. LeBlanc, K., Baranoski, S. “Prevention and Management of Skin Tears.” Advances in Skin & Wound Care 22(7):325-32, 2009.
74. LeBlanc, K. Skin Tear Prevalence, Incidence and Associated Risk Factors in the Long-term Care Population. Kingston, ON: Queen’s University. Theses (Queen’s University (Kingston, ON)), 2017. Available at: http://hdl.handle.net/1974/22678
75. Kaya, G., Saurat, J. Dermatoporosis: A Chronic Cutaneous Insufficiency/Fragility Syndrome. Dermatology 215(1):284, 2007.
76. Payne, R.L., Martin, M.C. “Defining and Classifying Skin Tears: Need for a Common Language…a Critique and Revision of the Payne-martin Classification System for Skin Tears.” Ostomy Wound Management 39(5):16, 1993.
77. Reddy, M. “Skin and Wound Care: Important Considerations in the Older Adult.” Advances in Skin & Wound Care 21(9):424-36, 2008.
78. Baharestani, M. “An Overview of Neonatal and Pediatric Wound Care Knowledge and Considerations.” Ostomy Wound Management 56(6):34, 2007.
79. Orstead, H., Campbell, K., Keast, D., et al. “Chronic Wound Caring…A Long Journey Towards Healing.” Ostomy Wound Management 47(10):26, 2001.
80. Reddy, M., Holroyd-Leduc, J., Cheung, C. Fundamentals of Geriatric Care in the Older “Chronic Wound Patient”. in D.L. Krasner, G. Rodeheaver, R. G. Sibbald, eds. Chronic Wound Care: A Clinical Sourcebook for Healthcare Professionals, 4th ed. Malvern, PA: HMP Communications, 2007:663.
81. Posthauer, M.E., Thomas, D.R. “Nutrition and Wound Care.” in S. Baranoski, E. Ayello, eds. Wound Care Essentials: Practice Principles. Springhouse, PA: Lippincott Williams & Wilkins, 2012.

82. Wysocki, A. “Anatomy and Physiology of Skin and Soft Tissue.” in R. Bryant, ed. Acute and Chronic Wounds: Nursing Management, 1st ed. London, UK: Mosby, 1992:1.
83. Pervin, L. “Polypharmacy and the Aging: Is There Cause for Concern?.” Gerontology Update: AJN 1(1): 10, 2008.
84. Tinette, M. “Clinical Practice: Preventing Falls in Elderly Persons.” The New England Journal of Medicine 348(42):49, 2003.
85. Benbow, M. “Skin Tears.” Journal of Community Nursing 23(1):14, 2009.
86. Bank, D., Nix, D. “Preventing Skin Tears in a Nursing and Rehabilitation Center: An Interdisciplinary Effort.” Ostomy Wound Management 52(9):38, 2006.
87. Baranoski, S. “How to Prevent and Manage Skin Tears.” Advances in Skin & Wound Care 16(5):268-70, 2003.
88. Baranoski, S. “Meeting the Challenge of Skin Tears.” Advances in Skin & Wound Care 18(2):74-5, 2005.
89. Neyens, J., Dijcks, B., Twisk, J., et al. “A Multifactorial Intervention for the Prevention of Falls in Psychogeriatric Nursing Home Patients, a Randomized Controlled Trial (RCT).” Age and Ageing 38(2):194, 2009.
90. Registered Nurses’ Association of Ontario (RNAO). Nursing Best Practice Guidelines: Prevention of Falls and Fall Injuries in the Older Adult, 2009. Retrieved March 23, 2013, from http://www.rnao.org/bestpractices/PDF/BPG_Falls_rev05.pdf
91. Birch, S., Coggins, T. “No-Rinse, One-Step Bed Bath: The Effects on the Occurrence of Skin Tears in a Long-Term Care Setting.” Ostomy/Wound Management 49:64-7, 2003.
92. Mason, S.R. “Type of Soap and the Incidence of Skin Tears Among Residents of a Long-Term Care Facility.” Ostomy Wound Management 43(8):26-30, 1997.
93. Brillhart, B. “Pressure Sore and Skin Tear Prevention and Treatment During a 10-Month Program.” Rehabilitation Nursing 30(3):85-91, 2005.
94. McTigue, T., D’Andrea, S., Doyle-Munoz, J., et al. “Efficacy of a Skin Tear Education Program. Improving the Knowledge of Nurses Practicing in Acute Care Settings.” Journal of Wound, Ostomy, and Continence Nursing 36(5):486-92, 2009.
95. Chaplain, V., Labreque, C., Woo, K., et al. French Canadian Translation and the Validity and Interrater Reliability of the ISTAP Skin Tear Classification System. Journal of Wound Care 27(9):S15-20, 2018.
96. Källman, U., LeBlanc, K., Bååth, C. Swedish Translation and Validation of the International Skin Tear Advisory Panel Skin Tear Classification System. International Wound Journal 16:13-8, 2019. doi: 10.1111/iwj.12975.
97. Skiveren, J., Bermark, S., LeBlanc, K., et al. Danish Translation and Validation of the International Skin Tear Advisory Panel Skin Tear Classification System. Journal of Wound Care 24(8):388, 2015.

98. Parisi, R., Symmons, D.P., Griffiths, C.E., et al. Global Epidemiology of Psoriasis: A Systematic Review of Incidence and Prevalence. Journal of Investigative Dermatology 133(2):377-85, 2013.
99. Brandon, A., Asfandyar, M., Sibbald, R.G. Diagnosis and Management of Cutaneous Psoriasis: A Review. Advances in Skin and Wound Care 32(2):58-69, 2019.
100. Krueger, G.G., Griffiths, C.E.M. Psoriasis: Epidemiology, Clinical Features, and Quality of Life. Annals of the Rheumatic Diseases 64(Suppl 2):ii18-23, 2005.
101. Langley, R.G.B., Krueger, G.G., Griffiths, C.E.M. Psoriasis: Epidemiology, Clinical Features, and Quality of Life. Annals of the Rheumatic Diseases 64(Suppl 2):ii18-23, 2005.
102. Brannon, H. June 20, 2019. Available at: https://www.verywellhealth.com/definition-of-auspitz-sign-bleeding-1069297. Accessed August 6, 2019.
103. Ladizinski, B., Lee, K.C., Wilmer, E., et al. A Review of Clinical Variants and the Management of Psoriasis. Advances in Skin & Wound Care 26(6):271-84, 2013.
104. Rodríguez-Zúñiga, M.J.M. Systematic Review and Meta-Analysis of the Association Between Psoriasis and Metabolic Syndrome. Journal of the American Academy of Dermatology 77(4):657-66.e8, 2017.
105. Li, X., Kong, L., Li, F., et al. Association Between Psoriasis and Chronic Obstructive Pulmonary Disease: A Systematic Review and Meta-Analysis. PLoS One 10(12):e0145221, 2015.
106. Candia, R., Ruiz, A., Torres-Robles, R., et al. Risk of Non-Alcoholic Fatty Liver Disease in Patients with Psoriasis: A Systematic Review and Meta-Analysis. Journal of the European Academy of Dermatology and Venereology 29(4):656-62, 2015.
107. Horreau, C., Pouplard, C., Brenaut, E., et al. Cardiovascular Morbidity and Mortality in Psoriasis and Psoriatic Arthritis: A Systematic Literature Review. Journal of the European Academy of Dermatology and Venereology 27(Suppl 3):12-29, 2013.
108. Denyer, J., Pillay, E., Clapham, J. Best Practice Guidelines for Skin and Wound Care in Epidermolysis Bullosa. An International Consensus. Wounds International, 2017.
109. Kirsner, R.S.D., Cassidy, S., Marsh, C., et al. Use of a Keratin-Based Wound Dressing in the Management of Wounds in a Patient with Recessive Dystrophic Epidermolysis Bullosa. Advances in Skin & Wound Care 25(9):400-3, 2012.
110. Pope, E., Lara-Corrales, I., Mellerio, J.E., et al.; for the EB International Consensus Panel. Epidermolysis Bullosa and Chronic Wounds: A Model for Wound Bed Preparation of Fragile Skin. Advances in Skin & Wound Care 26(4):177-88, 2013.

111. Pope, E., Lara-Corrales, I., Mellerio, J., et al. A Consensus Approach to Wound Care in Epidermolysis. Journal of the American Academy of Dermatology 67(5):904-17, 2012. doi: 10.1016/j.jaad.2012.01.016.
112. Trent, J.T., KIrsner, R.S. Epidermolysis Bullosa: Identification and Treatment. Advances in Skin & Wound Care 16(6):284,286,289-90, 2003.
113. Ayello, E.A., Levine, J.M., Langemo, D., et al. Reexamining the Literature on Terminal Ulcers, SCALE, Skin Failure, and Unavoidable Pressure Injuries. Advances in Skin & Wound Care 32(3):109-21, 2019.
114. Goode, P.S., Allman, R.M. "The Prevention and Management of Pressure Ulcers." The Medical Clinics of North America 73:1511-524, 1989.
115. LaPuma, L. "The Ethics of Pressure Ulcers." Decubitus 4(2):43-4, 1991.
116. Witkowski, J.A., Parish, L.C. "Skin Failure and the Pressure Ulcer." Decubitus 6(5):4, 1993.
117. Witkowski, J.A., Parish, L.C. The Decubitus Ulcer: Skin Failure and Destructive Behavior. International Journal of Dermatology 39(12):894-6, 2000.
118. Leijten, F.S., DeWeerd, A.W., Poortvliet, D.C., et al. "Critical Illness Polyneuropathy in Multiple Organ Dysfunction Syndrome and Weaning from the Ventilator." Intensive Care Medicine 22:856-61, 1996.
119. Hobbs, L., Spahn, J.G., Duncan, C. Skin Failure: What Happens When This Organ System Fails. Poster Presented at the WOCN Society 32nd Annual Conference, Toronto, Ontario, CA; June 2000.
120. Langemo, D.K., Brown, G. "Skin Fails Too: Acute, Chronic, and End-Stage Skin Failure." Advances in Skin & Wound Care 19:206-11, 2006.
121. Delmore, B., Cox, J., Rolnitzky, L., et al. Differentiating a Pressure Ulcer from Acute Skin Failure in the Adult Critical Care Patient. Advances in Skin & Wound Care 28(11):514-24, 2015.
122. Levine, J.M. Skin Failure: An Emerging Concept. Journal of the American Medical Directors Association 17(7):666-9, 2016.
123. Levine, J.M. Unavoidable Pressure Injuries, Terminal Ulceration, and Skin Failure: In Search of a Unifying Classification System. Advances in Skin & Wound Care 30(5):200-2, 2017.
124. Olshansky, K. Organ Failure, Hypoperfusion, and Pressure Ulcers Are Not the Same as Skin Failure: A Case for a New Definition. Advances in Skin & Wound Care 29(4):150, 2016.
125. White-Chu, E.F., Langemo, D. Skin Failure: Identifying and Managing an Under Recognized Condition. Annals of Long-Term Care 20(7):28-32, 2012.
126. Worley, C.A. Skin Failure: The Permissible Pressure Ulcer? Dermatology Nursing 19(4):384-5, 2007.
127. Alvarez, O., Brindle, C.T., Langemo, D., et al. The VCU Pressure Ulcer Summit: The Search for a Clearer Understanding and More Precise Clinical Definition of the Unavoidable Pressure Injury. Journal of Wound, Ostomy, and Continence Nursing 43(5):455-63, 2016.

128. Kennedy, K.L. The Prevalence of Pressure Ulcers in an Intermediate Care Facility. Decubitus 2(2):44-5, 1989.
141. Understanding the Kennedy Terminal Ulcer. Available at: www.kennedyterminalulcer.com. Last accessed December 11, 2018.
129. Brennan, M.B., Trombley, K. Kennedy Terminal Ulcers. A Palliative Care Units Experience Over a 12-Month Period of Time. World Council of Enterostomal Therapists Journal 30(3):20-2, 2010.
130. Trombley, K., Brennan, M.R.,Thomas, L., et al. Prelude to Death or Practice Failure? Trombley Brennan Terminal Tissue Injuries. The American Journal of Hospice & Palliative Care 29(7):541-5, 2012.
131. Sibbald, R.G., Krasner, D.L., Lutz, J.B., et al. Skin Changes at Life's End (SCALE): A Preliminary Consensus Statement. World Council of Enterostomal Therapists Journal 28(4):15-22, 2008.
132. Sibbald, R.G., Krasner, D.L. "SCALE: Skin Changes at Life's End." Wounds 21(12):329-36, 2009.
133. Sibbald, R.G., Krasner, D.L., Lutz, J. SCALE: Skin Changes at Life End: Final Consensus Statement: October 1, 2009. Advances in Skin & Wound Care 23(5):225-36, 2010.
134. Krasner, D.L., Stewart, T.P. SCALE Wounds: Unavoidable Pressure Injury. Wounds 27(4):92-4, 2015.
135. Brown, G. Long-term Outcomes of Full-Thickness Pressure Ulcers: Healing and Mortality. Ostomy Wound Management 49(10):42-50, 2003.
136. Miller, M.S. The Death of the Kennedy Terminal Ulcer. Journal of the American College of Clinical Wound Specialists 8(1-3):44-6, 2017.
137. Schank, J.E. The Kennedy Terminal Ulcer—alive and Well. Journal of the American College of Clinical Wound Specialists 8(1-3):54-5, 2016.
138. CentersforMedicare&MedicaidServices. CMSManualSystem.Pub100-07StateOperations Provider Certification. Transmittal 5. 2004. Available at: www.cms.gov/Regulations-and-Guidance/Guidance/Transmittals/downloads/R5SOM.pdf. Last accessed December 21, 2018.
139. Carlsson, M.E., Gunningberg, L. Predictors for Development of Pressure Ulcer in End-of-Life Care: A National Quality Register Study. Journal of Palliative Medicine 20(1):53-8, 2017.

14

Pressure Injuries

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Objectives

After completing this chapter, you'll be able to:

- explain the current etiology of a pressure injury
- discuss the significance of pressure injuries as a global and across care setting healthcare problem
- describe how to complete a risk assessment tool
- discuss strategies for pressure injury prevention
- define NPIAP pressure injury classification system
- discuss strategies for treating a patient with a pressure injury
- develop a plan of care to prevent and/or treat device-related pressure injury (also known as MDRPI)

- explain how to determine pressure injury prevalence and incidence.

Pressure Injuries as a Healthcare Concern

Despite the efforts of skin and wound care clinicians, pressure injuries continue to occur and thus require attention to prevention and treatment strategies based on the latest scientific evidence. Pressure injuries remain a global healthcare concern and require an interdisciplinary approach to ensure consistency in care and management.[1] While some believe that pressure injuries are a nurse-sensitive indicator, others believe the etiology of pressure injuries to be multifactorial and complex and therefore a healthcare system– or team-sensitive indicator.[2,3] All involved in the patient's care need to collaborate, communicate, and be responsible for the prevention and treatment of pressure injuries.[2,4]

Over the centuries, pressure injuries have been referred to as pressure ulcers, decubitus ulcers, bedsores, and pressure sores. The term *pressure injury* has become a preferred term in some parts of the world, particularly in the United States, Canada, and Australia, as it most closely describes the etiology and resultant tissue damage.[1] While in different parts of the world, tissue damage from sustained pressure and shear may be referred to with a term other than "pressure injury," it would be ideal to move towards consistent terminology and improved ICD-11 coding (although it will not be in effect for a few more years). Due to the combined efforts of the NPIAP and EPUAP, the terms pressure ulcer and pressure injury remain as synonyms in the current ICD-11 coding system, with a separate code added for deep tissue pressure injury (DTPI). (Personal J. Cuddigan, personal communication, 2020.) The 2019 pressure ulcer clinical guideline jointly compiled by the European Pressure Ulcer Advisory Panel (EPUAP), National Pressure Injury Advisory Panel (NPIAP), and the Pan Pacific Pressure Injury Alliance (PPPIA) released the following to serve as a common definition: *"A pressure injury is defined as localized damage to the skin and/or underlying tissue, as a result of pressure or pressure in combination with shear. Pressure injuries usually occur over a bony prominence but may also be related to a*

medical device or other object."[1] The sacrum, coccyx, hips, heels, and ears are the most common sites for the presence of pressure injuries (Fig. 14-1). Relying just on the *depth* of tissue damage rather than incorporating tissue type as well may be misleading as some pressure injuries occur in locations where there is little adipose tissue. An example of this is the ear where the injury may appear to be shallow but still extend through to the subcutaneous tissue or cartilage. Some damage is also present internally with skin that remains intact without breakdown, as observed in deep tissue injuries cases[1] (Box 14-1).

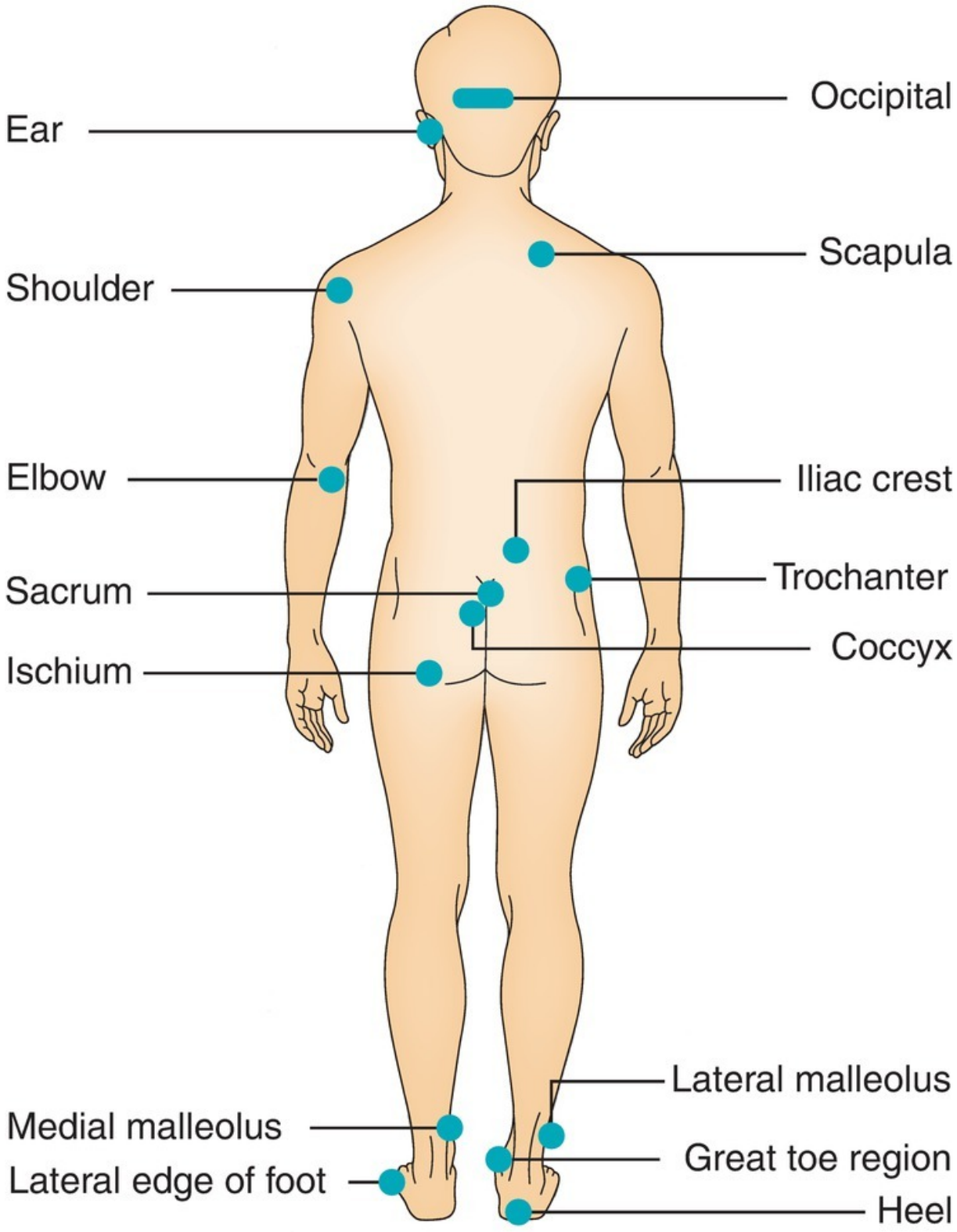
Occipital
Ear
Scapula
Shoulder
Elbow
Iliac crest
Trochanter
Sacrum
Coccyx
Ischium
Lateral malleolus
Medial malleolus
Great toe region
Lateral edge of foot
Heel

Figure 14-1. Pressure injury sites. Shown in this illustration are the most common sites where pressure injuries develop.

BOX 14-1 NPIAP Pressure Injury Classification System April 2016

Stage 1: Pressure Injury: Nonblanchable Erythema of Intact Skin

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- Intact skin with a localized area of nonblanchable erythema that may appear differently in darkly pigmented skin.
- Presence of blanchable erythema or changes in sensation, temperature, or firmness may precede visual changes.
- Color changes do not include purple or maroon discoloration; these may indicate DTPI.

Stage 2: Pressure Injury: Partial-Thickness Skin Loss with Exposed Dermis

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- Partial-thickness loss of skin with exposed dermis. The wound bed is viable, pink or red, moist and may also present as an intact or ruptured serum-filled blister. Adipose (fat) is not visible, and deeper tissues are not visible. Granulation tissue, slough, and eschar are not present. These injuries commonly result from adverse microclimate and shear in the skin over the pelvis and shear in the heel. This stage should not be used to describe moisture-associated skin damage (MASD) including incontinence-associated dermatitis (IAD) intertriginous dermatitis (ITD), medical adhesive–related skin injury (MARSI), or traumatic wounds (skin tears, burns, abrasions).

Stage 3: Pressure Injury: Full-Thickness Skin Loss

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- Full-thickness tissue loss of skin, in which adipose (fat) is visible in the ulcer and granulation tissue and epibole (rolled wound edges) are often present. Slough and/or eschar may be visible. The depth of tissue damage varies by anatomical location; areas of significant adiposity can develop deep wounds. Undermining and tunneling may occur. Fascia, muscle, tendon, ligament, cartilage, and/or bone are not exposed. If slough or eschar obscures the extent of tissue loss this is an unstageable pressure injury.

Stage 4: Pressure Injury: Full-Thickness Skin and Tissue Loss

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- Full-thickness skin and tissue loss with exposed or directly palpable fascia, muscle, tendon, ligament, cartilage, or bone in the ulcer. Slough and/or eschar may be visible. Epibole (rolled edges), undermining and/or tunneling often occurs. Depth varies by anatomical location. If slough or eschar obscures the extent of tissue loss, this is an unstageable pressure injury.
- Unstageable pressure injury: Obscured full-thickness skin and tissue loss

© Elizabeth A. Ayello

- Full-thickness skin and tissue loss in which the extent of tissue damage within the ulcer cannot be confirmed because it is obscured by slough or eschar. If slough or eschar is removed, a stage 3 or stage 4 pressure injury will be revealed. Stable eschar (i.e., dry, adherent, intact without erythema or fluctuance) on the heel or ischemic limb should not be softened or removed.

Deep Tissue Pressure Injury: Persistent Nonblanchable Deep Red, Maroon, or Purple Discoloration

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Intact or nonintact skin with localized area of persistent nonblanchable deep red, maroon, purple discoloration or epidermal separation revealing a dark wound bed or blood-filled blister. Pain and temperature change often precede skin color changes. Discoloration may appear differently in darkly pigmented skin. This injury results from intense and/or prolonged pressure and shear forces at the bone–muscle interface. The wound may evolve rapidly to reveal the actual extent of tissue injury, or may resolve without tissue loss. If necrotic tissue, subcutaneous tissue, granulation tissue, fascia, muscle, or other underlying structures are visible, this indicates a full-thickness pressure injury (Unstageable, Stage 3, Stage 4). Do not use DTPI to describe vascular, traumatic, neuropathic, or dermatologic conditions.

Edsberg, L.E., Black, J.M., Goldberg, M., et al. Revised National Pressure Ulcer Advisory Panel Pressure Injury Staging System: Revised Pressure Injury Staging System. *Journal of Wound, Ostomy, and Continence Nursing* 43(6):585-97, 2016.

The true extent of incidence and prevalence of pressure injuries is not fully known and hard to effectively compare between settings, as it is not a consistently reported-on event across all healthcare settings in the world. Additionally, there is significant variation in methodology, frequency, and rigor of data collection to support truly accurate data comparison among settings. The 2019 EPUAP NPIAP PPPIA clinical guideline addressed this issue and provides structured guidance for healthcare providers to determine prevalence, incidence, and facility-acquired rates in a correct and consistent manner.[1] This guide on data collection methodology includes a set of approved formulas recommended for use in incidence and prevalence data collection projects. This has the potential to positively impact data generation quality, accuracy, and comparability of incidence and prevalence figures across all healthcare settings adopting this methodology.

We do know, however, that prevalence and incidence figures are significant enough to warrant national healthcare improvement initiatives in the United States and other countries. As early as 1989, the US federal government focused its attention on pressure injuries with the appointment of a panel charged with developing the Agency for Health Care Policy and Research (AHCPR) guidelines.[5,6] Since

that time, clinical practice guidelines regarding pressure injuries have been released by several other organizations such as the Wound, Ostomy and Continence Nurses Society (WOCN),[7] the Registered Nurses Association of Ontario (RNAO),[8] the Wound Healing Society (WHS),[9] and the Canadian Association of Wound Care.[10]

Padula and Delarmente[11] reported that US spending in 2018 on hospital-acquired pressure injuries (HAPI) is $26.8 billion, with Medicare spending on pressure injuries $22 billion. “About 59% of these costs are disproportionately attributable to a small rate of Stages 3 and 4 full-thickness wounds, which occupy clinician time and hospital resources. Hospital acquired pressure injuries (HAPIs) remain a concern with regard to hospital quality in addition to being a major source of economic burden on the US health care system.”[11]

The 2019 EPUAP/NPIAP/PPPIA evidence-based Clinical Practice Guideline[1] consists of five main sections:

- background materials including etiology
- needs of special populations
- prevention of pressure injuries
- topics relevant to prevention and treatment of pressure injuries
- Treatment of pressure injuries and implementing the guideline.

The guideline includes 115 evidence-based recommendations and 61 good practice statements. New in this edition are a comprehensive explanation of the etiological factors and damage cascade ranging from microscopic (cell-scale) damage up to the clinically identifiable injury; specific implementation considerations; and 20 quality indicators that can be used to help in implementation of this guideline.[1]

In the EPUAP/NPIAP/PPPIA Clinical Practice Guideline, there are seven recommendations at the A level of evidence strength (Box 14-2).[1] Wound care interventions and modalities have often been based on an “it works for me” attitude. In the guideline there is an indication of the strength of each recommendation. Based on consensus voting by the experts who developed the guideline, a system using arrows up, down, or neutral is given to indicate how confident a healthcare

professional can be that implementing the recommendation will do more good than harm.[1] (See Appendix "Definitions of the EPUAP NPIAP PPPIA 2019 Clinical Guideline Strengths of Evidence and Strengths of Recommendations."[1]) We need to encourage healthcare providers to participate in research studies with the aim to generate more evidence in the future to direct and improve our clinical decision-making process, thereby improving patient outcomes.

BOX 14-2 Clinical Recommendations: A Level of Evidence

1.1Consider individuals with limited mobility, limited activity and a high potential for friction and shear to be at risk of pressure injuries. Strength of recommendation ↑↑

1.2Consider individuals with a Category/Stage I pressure injury to be at risk of developing a Category/Stage II or greater pressure injury. ↑↑

1.7Consider the impact of diabetes mellitus on the risk of pressure injuries. ↑↑

2.2Inspect the skin of individuals at risk of pressure injuries to identify presence of erythema. ↑↑

17.1Administer pulsed current electrical stimulation to facilitate wound healing in recalcitrant category/stage 2 pressure injuries and category/stage 3 or 4 pressure injuries ↑

20.5At an organizational level, develop and implement a structured, tailored and multifaceted quality improvement program to reduce the incidence of pressure injuries. ↑↑

20.10At a professional level, provide education in pressure injury prevention and treatment as part of a quality improvement plan to reduce the incidence of pressure injuries. ↑↑

European Pressure Ulcer Advisory Panel, National Pressure Injury Advisory Panel and Pan Pacific Pressure Injury Alliance. In: E. Haesler, ed. *Prevention and Treatment of Pressure Injuries/Injuries: Clinical Practice Guideline. The International Guideline 2019*. EPUAP/NPIAP/PPPIA, 2019[1].

Wound Etiology

How do pressure injuries occur? This is an interesting and challenging question, of which the majority of scientific knowledge has been formed in the last two decades through studies on the development of new bioengineering fields and methods in the fields of mechanobiology, tissue engineering, and live cell imaging. The 2019 international EPUAP/NPIAP/PPPIA clinical practice guideline explains that “tissue damage occurs as a result of intense and/or prolonged exposure to sustained deformation in compression (perpendicular to the tissue surface), tension or shear (parallel to tissue surface), or a combination of these loading modes. The tolerance of soft tissue for sustained deformations differs by tissue type and may also be affected by microclimate, perfusion, age, health status (chronic or acute), comorbidities, and conditions of the soft tissues.”[1]

Based on the work of Coleman and colleagues[12] the 2019 International EPUAP/NPIAP/PPPIA guideline states that “the relevant risk factors for pressure injuries are present in two groups:

1. Mechanical boundary conditions, including magnitudes and time durations of the applied mechanical loads, and their mode of action, such as compressive or shear.
2. Tolerance of the individual (internal anatomy including the prominence of bony structures, tissue morphology, mechanical properties of tissues, tissue repair capacity, and transport and thermal properties of tissues).

The internal deformations, strains and stresses within soft tissues, the function of the immune system, quality of vascular perfusion and lymphatic drainage under the applied loads, and the individual tolerance to the tissue loads and associated biochemical conditions are dictated by complex interactions between the factors in the above two groups. Acting in unison, the two factor groups determine the time taken for a pressure injury to develop in the individual, as well as the extent and severity to which it will develop if that individual or an affected body part of the individual is immobile and/or insensate.”[1]

Simply stated, it is how the soft tissue of an individual responds physiologically to the sustained exposure of mechanical loading.

Practice Point

Sustained deformation can damage tissue cells directly within minutes by disrupting cell structures between cells and their surroundings.

Sustained deformation can cause ischemia, which can damage tissue cells in hours.

Sustained deformation can lead to inflammatory edema, which can damage tissues through increase in interstitial pressures that deforms additional cells and may obstruct vascular perfusion and lymphatic flow.Individual body characteristics, including the sharpness of bony prominences and soft tissue mass and composition, affect the deformation and mechanical stress states in tissues.

Deformation tolerance depends on individual anatomical, physiological, and biomechanical factors.

Sustained tissue deformations can be alleviated with the aid of effective prevention interventions that absorb a sufficient portion of the loads, thereby reducing tissue exposure to the sustained loading.

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The Etiology Chapter of the 2019 EPUAP/NPIAP/PPPIA Guideline is a comprehensive compilation of the contemporary scientific knowledge on pressure injury formation. The essence of this etiological description is provided here; however, readers should refer to the original publication for the detailed and complete scientific explanations of the etiology of pressure injuries.[1]

Briefly, forces acting on the surface of the sedentary body (in lying or sitting positions) are not directly translated to forces on internal tissue structures or the microscale of cells. The internal distribution of forces, deformations, and mechanical stresses in the body tissues and their microarchitecture depend on the internal anatomy and the mechanical properties of the tissues at multidimensional scales. These are always characteristic to the individual, and also progressively change over time and are affected by diseases or chronic conditions. This is the primary reason for the unfeasibility of predicting the timing and extent of pressure injuries in individual patients based solely on measurements taken at the surface of the body, for example interface pressure maps or skin temperature distribution data.

While it has been known since the early days of biomechanical research that bodyweight loads may be distributed in distinguishable patterns in the bodies and tissues of different persons, the consequences of these loading exposures on cell and tissue viability and biological function have only been clarified recently. This latest mechanobiological research (described in detail in the 2019 Guideline publication) demonstrated that sustained exposure to tissue deformations causes gradual degradation of the cytoskeleton and plasma membrane of the distorted cells within the loaded tissues. This leads, within short times in the order of tens of minutes, to impaired control of the distorted cells over biomolecular transport through their plasma membranes, and therefore, to rapid loss of the homeostasis of the distorted cells. Importantly, the loss of control over transport in the affected cells is caused directly by the sustained exposure of these cells and tissues to mechanical loading; it occurs even under nonischemic conditions, and it is *not* related to the availability of oxygen, glucose or other metabolites transported by the blood circulation. That is, pressure injuries are caused first and foremost by sustained tissue distortions and deformations, and not primarily by poor blood supply.

In fact, ischemic conditions in the distorted tissues develop much later in the damage cascade (if no effective intervention has been taken to stop the tissue damage from progressing). These ischemic effects are also triggered by the death of the first cells due to the sustained deformation exposures, as explained above. Specifically, with the deformation-inflicted death of the first cells, as described

above, an inflammatory response is triggered, by secretion of chemokines and neurotransmitters (e.g., histamine) from the damaged/dying cells, and from nearby immune cells. These signaling molecules cause blood vessel dilation and increased permeability of the vascular walls, to allow the crossing (extravasation) and then the migration of additional immune cells (e.g., phagocytes) from the blood circulation to the tissue damage site (from which the molecular alarm signals have been sent). The dilation of vessels near the damage site and their increased permeability leads to ongoing leakage of plasma fluids into the interstitial spaces, which gradually forms edema. If, for some reason, the soft tissues at that location cannot freely expand in volume (i.e., swell) or if tissues are limited in doing so, for example if these tissues are confined between bony structures (e.g., the sacrum) and a support surface (e.g., a mattress), then interstitial pressures would rise rapidly and cause additional cell-deformation damage (again by exposure to sustained distortions). Furthermore, the rise in interstitial pressures will lead to obstruction, or even possible localized occlusions of vascular and lymphatic vessels. This additional mechanical loading, caused by the escalating edema, contributes to ischemia and the resulting ischemic metabolic stress.

While this new scientific knowledge has shifted the etiological paradigm from an exclusive focus on ischemia to a more holistic scope, highlighting the fundamental role of tissue deformation exposures in the injury spiral, it also created new opportunities for prevention, early detection, and overall, technology-aided medicine, which were lacking in the field before. One good example is the novel technology for measuring tissue biocapacitance, which is a biophysical marker for detecting initial tissue damage, based on the increased level of fluids that build up in the interstitial space as the inflammatory response to the deformation-inflicted cell death kicks in. The greater interstitial fluid contents due to the leaky vessels will increase the biocapacitance of the affected tissues (water has a greater dielectric constant than tissue proteins; the larger the effective dielectric constant of tissues, the greater the biocapacitance). The early detection of interstitial edema resulting from tissue inflammation might be detected by measurements of tissue biocapacitance, suggesting a potential future tool for early detection of pressure injuries.

Body tissues differ in their ability to tolerate mechanical loads. The blood supply to the skin originates in the underlying muscle and subdermal adipose tissues. Muscle is more sensitive to deformation-inflicted damage than skin tissue.[1] Tissue tolerance is further compromised by extrinsic and intrinsic factors. Extrinsic factors that are gaining momentum as to the role they may play in pressure injury development include moisture and temperature and are usually collectively referred to as microclimate.[1,12] EPUAP/NPIAP/PPPIA[1] guideline suggests that microclimate plays a role in the development of superficial (categories 1 and 2) pressure injuries. They also suggest that another intrinsic factor, tissue perfusion (or lack thereof), and the resulting ischemia may be another important part of explaining how pressure injuries occur.[1] A 2013 comprehensive systematic review of risk factor research by Coleman and colleagues helps explain the complex interaction between increased pressure intensity/duration and the extrinsic and intrinsic factors that affect tissue tolerance to pressure.[1,13] Based on their review of 54 studies of 34,449 patients in both acute care and the community, they identified three primary independent predictors for pressure injury development: mobility/activity, perfusion (including diabetes), and skin/pressure injury status.[13] Activity and mobility limitations create the necessary conditions for pressure injuries to develop (i.e., unrelieved pressure). Individuals who are bedbound, chairfast, and unable to effectively reposition themselves fall into these risk factor categories and should be considered at risk. Epidemiological studies show that limitations in activity and mobility are independently predictive of pressure injuries. Changes in sensory perception may further impair movement.

The 2019 EPUAP/NPIAP/PPPIA Guideline updates the systematic review completed by Coleman and colleagues in 2013 using the same methodology for reviewing the epidemiological evidence. Limited mobility, limited activity, and high potential for friction and shear have the strongest supporting evidence (A). In fact, limited mobility is a necessary condition for pressure injury development. There is a slight reordering of the strength of evidence for risk factors that contribute to pressure injury development. Stage 1 pressure injury and diabetes mellitus each carry an A level strength of evidence; these factors are indicative of impaired tissue tolerance and contribute to pressure injury

development in the presence of immobility. Factors supported by a B1 level of evidence include perfusion and circulation deficits and increased body temperature. C level evidence supports existing pressure injury, oxygenation deficits, nutritional status, moist skin, older age, impaired sensory perception, and selected blood tests as contributing risk factors. Good Practice Statements (GPS) based on expert opinion encourage clinicians to consider the following factors in their risk assessments—previous pressure injuries (especially stages 3 and 4), other skin alterations, pain at pressure points, and deficits in general health status. Special populations such as those undergoing operative procedures, critically ill individuals, neonates, and children have unique risk factors that should be also be assessed.

In keeping with the premise of assessing the whole person for pressure injury risk, patient comorbidities have also been studied. Two studies have found that comorbidities such as diabetes mellitus and vascular disease are predictors of heel pressure injury risk in hospitalized adult patients.[14,15]

Once the conditions for increased pressure intensity and duration are established, intrinsic and extrinsic factors affecting tissue tolerance contribute to pressure injury development. According to epidemiological evidence, these factors fall under several categories, which include factors affecting perfusion and oxygenation (e.g., hypotension, hemodynamic instability, peripheral vascular disease, diabetes, vasopressor drugs, need for supplemental oxygen); poor nutritional status (e.g., decreased intake of nutrients—especially protein, weight loss, low albumin); and skin moisture (e.g., urinary or fecal incontinence, excessive sweating, or wound drainage).[1]

Shear has the potential to damage deeper tissue. Shear and friction are two physically separate, but strongly coupled phenomena. Together they contribute to create cell deformation damage leading to inflammation, tissue ischemia, and eventually pressure injury development.

Frictional forces act parallel to the skin surface and may damage the epidermis causing friction blisters but not pressure injuries.[1] The tissue injury resulting from friction may look like a superficial skin insult. With repetitive frictional forces on fleshy (nonbony prominence) parts

of the body (e.g., repeatedly sliding down in a recliner or chair), the skin may become rough or hyperkeratotic with scaling, lichenification, and discoloration.[16,17] Frictional forces may translate to deeper tissue distortions that create internal tissue shear.

Shear (shear stress) is “the force per unit area exerted parallel to the perpendicular plane of interest”[1] while shear strain is the “distortion or deformation of tissue as a result of shear stress.”[1] You can think of shear stress and strain as pulling the bones of the pelvis in one direction and the skin in the opposite direction (Fig. 14-2). The deeper fascia slides downward with the bone, while the superficial fascia remains attached to the dermis. This primarily distorts cell structures and induces deformation-inflicted cell damage leading to an inflammatory response being triggered.[18,19] The ultimate result is massive cellular death and tissue necrosis (Fig. 14-3).

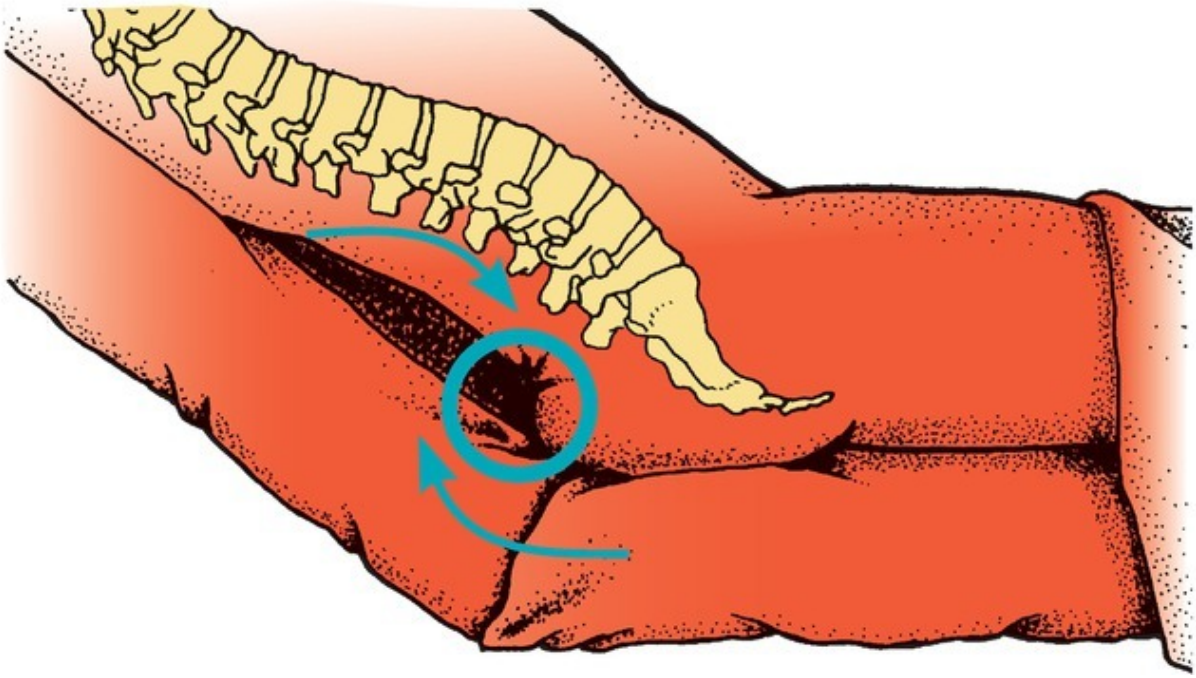


Figure 14-2. Shearing force. Shear injury is a mechanical force parallel, rather than perpendicular, to an area of tissue. In this illustration, gravity pulls the body down the incline of the bed. The skeleton and

attached deep fascia slide within the skin, while the skin and superficial fascia, attached to the dermis, remain stationary, held in place by friction between the skin and the bed linen. This internal slide compromises blood supply to the area and deforms or distorts tissue.

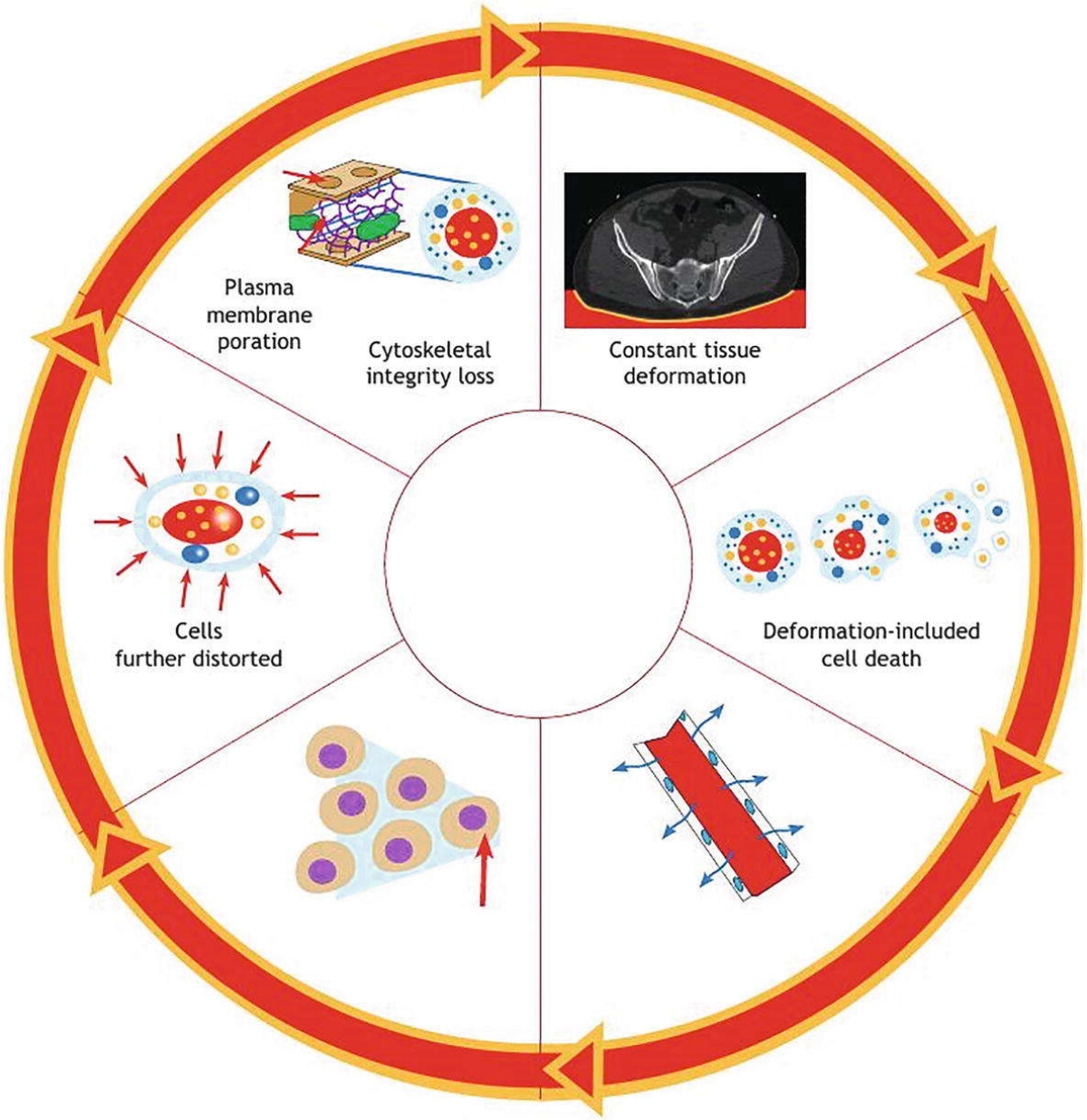


Figure 14-3. Etiology of pressure injuries. (From Amit © A. Gefen, used with permission).

Practice Point

Shear injury is initially not clinically visible at the skin level as it occurs underneath the skin in the deeper layers of subcutaneous tissue.

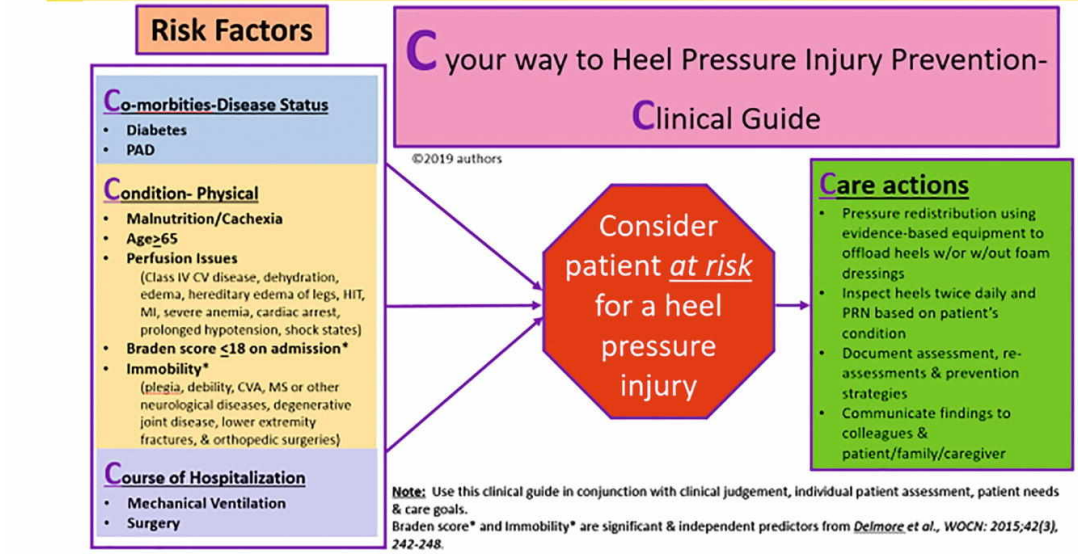
Friction injury on the other hand is clearly visible on the skin surface presenting with erythema, sometimes blistering, and often epidermal skin loss but not pressure injuries.

Elevation of the head of the bed higher than 30 degrees increases the likelihood of shear injury in the deep soft tissues present in the spine and hip juncture and may account for the number of sacral ulcers seen in practice.

Several national surveys demonstrated the most common site for pressure injuries among patients in acute care facilities to be the sacrum, followed by the heels.[20] The incidence of heel injuries has increased incrementally over the past decade, creating a need for prevention protocols targeting the heels. The HEELS© mnemonic[21] can be used to care for heels at risk for pressure injuries (Box 14-3).

BOX 14-3 Heel Pressure Injury Prevention Clinical Enabler

Heel Pressure Injury Prevention Clinical Enabler



Delmore, B, Ayello, EA, Smith, D, Rolnitzky L, Chu AS. Refining heel pressure injury risk factors in the hospitalized patient. Advances in Skin and Wound Care. 32(11): 512-9.

© Delmore, Ayello & Smart 2019

Device-Related Pressure Injuries

A growing clinical concern is that of pressure injuries caused by either medical devices or other objects attached to the skin. Device-related pressure injuries are known as DRPI while medical device–related pressure injuries is abbreviated as MDRPI. Both terms are interchangeably used (Fig. 14-4A and B). In the survey of 86,932 patients in acute care facilities from the International Pressure Ulcer Prevalence (IPUP) data, of all pressure injuries identified (17,911), 9.1% (1,631) were device related, with the ear being the most common site (20%).[18] A more recent retrospective analysis of IPUP data of 99,876 patients (all care settings) found that there were 11,651 pressure injuries with 6.9% (804) being MDRPI. Once again, ears (27%) were the most common anatomical site for MDRPI.[22] Kayser et al. also reported that facility-acquired MDRPIs formed 3 days faster than did non-MDRPIs.[22] MDRPIs were usually superficial as 58% were stage 1 (28%) or stage 2 (30%).[22] This supports a decrease in reported rates of stage 3 MDPRI (7.5%) in the Kayser et al. study compared to earlier rates from the Minnesota Safe Skin hospital initiative rates of 63% of MDRPI where most (74%) of MDRPI were not discovered till stage 3.[23] Patients with medical devices are 2.4 times more likely to develop any pressure injury.[24]

Figure 14-4. (A) Device-related pressure injury from a compression stocking. (© J. M. Levine, MD, used with

permission). **(B)** Device-related pressure injury from a tracheostomy tube. (© J. M. Levine, MD, used with permission).

Devices most commonly associated with pressure injuries include respiratory devices such as oxygen tubing (ears), nasotracheal tubes (ET, mouth and lips), nasogastric tubes, continuous positive airway pressure (CPAP) masks, and biphasic positive airway pressure masks over the bridge of the nose and face (Bi-PAP) and orthopedic devices including cervical collars (neck and head), halo devices, external fixators, and plaster casts. Other medical devices includes splints, braces, urinary catheters, stoma supplies and fecal containment devices, venous or dialysis catheters, heel suspension devices, thromboembolic deterrent hose, restraints, graduated compression stockings, and intermittent pneumatic compression device sleeves (lower extremities).[1] It is generally accepted that any tube, electrode, sensor, or other rigid or stiff device element under pressure can create pressure damage. Edematous patients are at particularly high risk and require a high degree of vigilance in the presence of attached devices.[24] Pressure injuries from medical devices present on mucosa are not staged in the NPIAP Pressure Injury Classification System. A position paper and helpful posters to help in educating clinicians about mucosal pressure injuries can be found on the NPIAP Web site (Fig. 14-5).[25] NPIAP posters available on their Web site (www.npiap.com) as well as the Sore© mnemonic[26] can be used to raise awareness of MDRPI/DRPI as a clinical concern (Fig. 14-6).

Figure 14-5. Mucosal pressure injury.

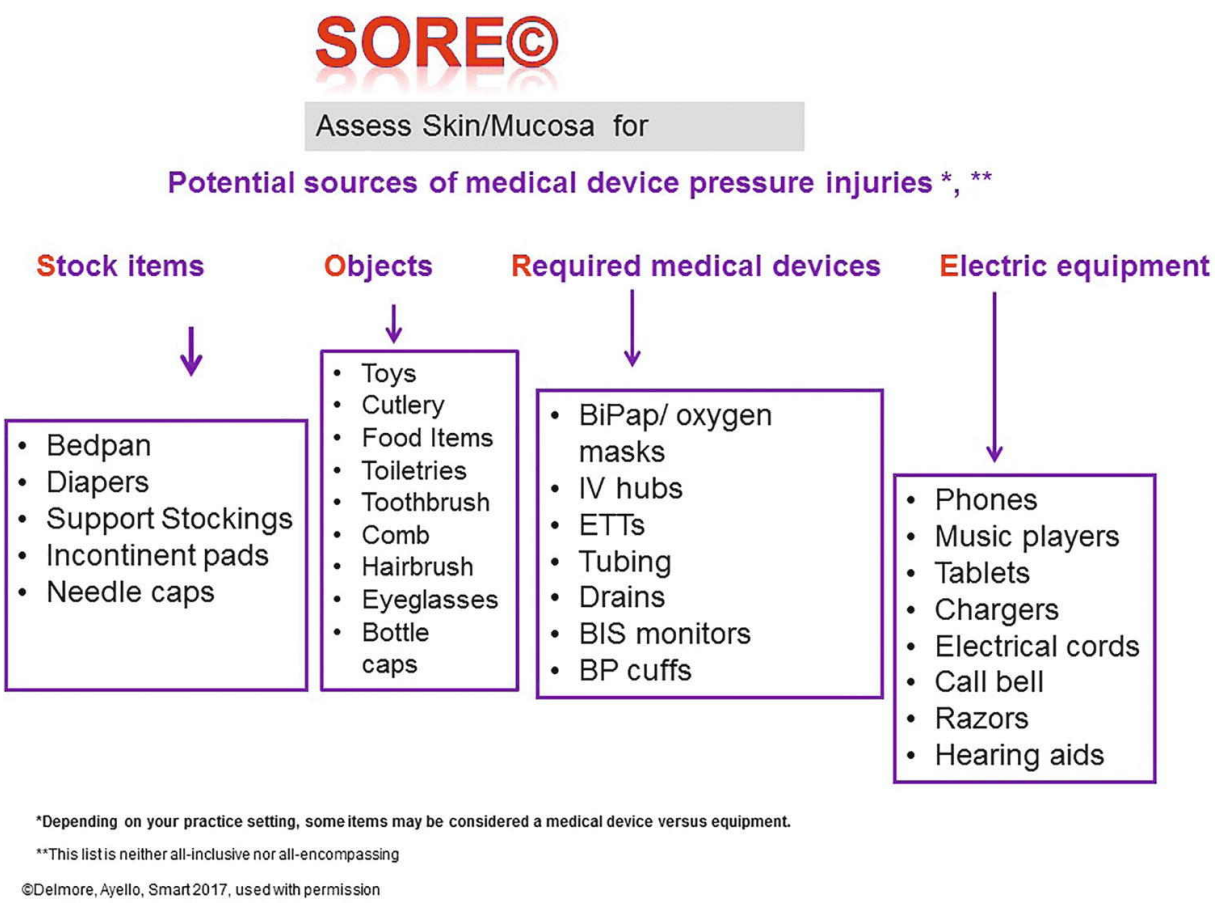


Figure 14-6. SORE©. (© Delmore, Ayello, Smart2017, used with permission).

Practice Point

Careful observation of the sacrum/coccyx, heels, and ears is warranted because these are the most frequent sites of pressure injuries.

Prevention

The prevention of pressure injuries is of vital importance. Elements of pressure injury prevention include identifying individuals at risk for developing pressure injuries, preserving skin integrity, treating the underlying causes of the pressure injury, relieving pressure and shear forces, paying attention to the total condition of the patient to correct any deficiencies, and educating the patient and his or her family about pressure injuries.

Risk Factors and Risk Assessment

Risk assessment is used to identify patients at risk and determine the type of risk that is present. Identifying individuals at risk for pressure injuries enables clinicians to make decisions about when to initiate preventive measures. This is an important decision that facilitates the timely utilization of resources, as the type of risk identified serves to guide the intensity and cost of preventive interventions to be implemented.[1]

Risk assessment is a comprehensive process that has now expanded beyond the most well-known and previously validated pressure injury risk assessment tools such as the Norton Scale,[27] Braden Scale,[28] and Waterlow Scale.[29] The guideline included several other useful risk assessment tools such as the Cubbin-Jackson Scale, SCIPUS, and Braden Q.[1] The EPUAP/NPIAP/PPPIA clinical guideline emphasizes that these risk assessment tools are to be used as part of a systematic and complete assessment for pressure injury risk and not as a stand-alone action.[1] The decision on which scale to use in a healthcare setting can be challenging as only one scale should be used per specific setting to prevent confusion on number interpretation. Reviewing the reliability (consistency) and validity (accuracy) of each scale should always be the first step in the decision-making process. Reliability for risk assessment scales is usually described in terms of interrater reliability. According to Ayello and Braden,[30] "a common measure of interrater reliability for a risk assessment tool is percentage agreement, which looks at the percentage of instances in which different raters assign the same score to the same patients. Validity, or accuracy, is measured by the ability of the tool to correctly predict who will or won't develop a pressure injury." Chou and colleagues identified over 747 full text articles and, based on 120 studies, eventually included in their review that while there was no difference among the instruments in diagnostic accuracy, the Braden, Norton, and Waterlow Scales did identify patients at increased risk but were weak predictors.[31] Furthermore, they urged more research to better understand how these tools compared with clinical judgment regarding pressure injury incidence. A 2014 meta-analysis by García-Fernández

and colleagues of risk assessment tools has provided new insights regarding the clinical applicability of risk assessment tools.[32]

Predictive validity is dependent on the sensitivity and specificity of the tool. Sensitivity is defined as "the percentage of individuals who develop a pressure injury who were assessed as being at risk for a pressure injury." A tool has good sensitivity if it correctly identifies true positives while minimizing false negatives. Specificity is defined as "the percentage of individuals who don't develop a pressure injury who were assessed as being not at risk for developing an injury." A tool has good specificity if it identifies true negatives and minimizes false positives."[30,33] Because of the amount of clinical research supporting their reliability and validity, the Norton,[27] Braden,[28] and Waterlow[29] scales are primarily used in clinical practice to determine pressure injury risk assessment.

Braden Scale

The Braden Scale is the most commonly used pressure injury assessment tool in the United States. Available in many languages (English, French, Portuguese)[34–36] and used worldwide, this copyrighted tool was created in 1987 by Barbara Braden and Nancy Bergstrom[28] and is available at http://www.bradenscale.com/images/bradenscale.pdf.[34] The Braden Scale has six subscales: sensory perception, moisture, activity, mobility, nutrition, and friction/shear[28,30,33] (Table 14-1). The scale is based on the two primary etiologic factors of pressure injury development—intensity and duration of pressure and tissue tolerance for pressure. "Sensory perception, mobility, and activity address clinical situations that predispose a patient to intense and prolonged pressure, while moisture, nutrition, and friction/shear address clinical situations that alter tissue tolerance for pressure."[30]

Table 14-1 Braden Scale: Predicting Pressure Ulcer Risk

Sensory Perception Ability to respond meaningfully to pressure-related discomfort	1. Completely limited: Unresponsive (doesn't moan, flinch, or grasp) to painful stimuli due to diminished level of consciousness or sedation OR limited ability to feel pain over most of body surface	2. Very limited: Responds only to painful stimuli. Can't communicate discomfort except by moaning or restlessness OR has a sensory impairment that limits the ability to feel pain or discomfort over half of body	3. Slightly limited: Responds to verbal commands but can't always communicate discomfort or need to be turned OR has some sensory impairment that limits ability to feel pain or discomfort in one or two extremities	4. No impairment: Responds to verbal commands. Has no sensory deficit that would limit ability to feel or voice pain or discomfort
Moisture Degree to which skin is exposed to moisture	1. Constantly moist: Skin is kept moist almost constantly by perspiration or urine. Dampness is detected every time patient is moved or turned.	2. Often moist: Skin is often but not always moist. Linen must be changed at least once per shift.	3. Occasionally moist: Skin is occasionally moist, requiring an extra linen change approximately once per day.	4. Rarely moist: Skin is usually dry; linen only requires changing at routine intervals.
Activity Degree of physical activity	1. Bedfast: Confined to bed.	2. Confined to chair: Ability to walk severely limited or nonexistent. Can't bear own weight and must be assisted into chair or wheelchair	3. Walks occasionally: Walks occasionally during day, but for very short distances, with or without assistance; spends majority of each shift in bed or chair	4. Walks frequently: Walks outside the room at least twice per day and inside room at least once every 2 h during waking hours
Mobility Ability to change and control body position	1. Completely immobile: Doesn't make even slight changes in body or extremity position without assistance	2. Very limited: Makes occasional slight changes in body or extremity position but unable to make frequent or significant changes independently	3. Slightly limited: Makes frequent though slight changes in body or extremity position independently	4. No limitations: Makes major and frequent changes in position without assistance

Nutrition Usual food intake pattern NPO: Nothing by mouth IV: Intravenously TPN: Total parenteral nutrition	1. Very poor: Never eats a complete meal. Rarely eats more than one-third of any food offered. Eats two servings or less of protein (meat or dairy products) per day. Takes fluids poorly. Doesn't take a liquid dietary supplement OR is NPO or maintained on clear liquids or IV fluids for more than 5 d	2. Probably inadequate: Rarely eats a complete meal and generally eats only about half of any food offered. Protein intake includes only three servings of meat or dairy products per day. Occasionally will take a dietary supplement OR receives less than optimum amount of liquid diet or tube feeding	3. Adequate: Eats over half of most meals. Eats a total of four servings of protein (meat, dairy products) each day. Occasionally will refuse a meal, but will usually take a supplement if offered OR is on a tube feeding or TPN regimen that probably meets most nutritional needs	4. Excellent: Eats most of every meal and never refuses a meal. Usually eats a total of four servings of meat and dairy products. Occasionally eats between meals. Doesn't require supplementation
Friction and Shear	1. Problem: Requires moderate to maximum assistance in moving. Complete lifting without sliding against sheets is impossible. Frequently slides down in bed or chair, requiring frequent repositioning with maximum assistance. Spasticity, contractures, or agitation leads to almost constant friction.	2. Potential problem: Moves feebly or requires minimum assistance. During a move, skin probably slides to some extent against sheets, chair, restraints, or other devices. Maintains relatively good position in chair or bed most of the time but occasionally slides down	3. No apparent problem: Moves in bed and in chair independently and has sufficient muscle strength to lift up completely during move. Maintains good position in bed or chair at all times	

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Each subscale contains a numerical range of scores, with 1 being the lowest score possible.[28] The sensory perception, moisture, activity, mobility, and nutrition subscales have scores ranging from 1 to 4. Friction/shear is the only subscale in which scores range from 1 to 3. Definitions of each subscale as to what patient characteristics to evaluate are given for each numerical ranking. While in the past emphasis was on the overall Braden Scale score, which was derived from totaling the numerical ratings from each of the six subscales, the 2019 EPUAP/NPIAP/PPPIA Clinical Guideline now recommends that the subscale scores and other patient risk factors not captured on risk

assessment scales should be included as part of an individualized pressure injury prevention plan of care.[1]

EPUAP/NPIAP/PPPIA Clinical Guideline[1] stress the importance of using a systematic process for determining pressure injury risk that may include clinical judgment, skin assessment, and review of risk factors not part of validated pressure injury risk assessment tools and not just relying on interpreting the total Braden Scale score because not all risk factors are quantified on the scale (low subscale scores need to be considered). The systematic review of patient risk factors for pressure injury development by Coleman and colleagues[13] also underscores the importance of a comprehensive risk assessment when they state “overall there is no single factor which can explain pressure injury risk, rather a complex interplay of factors which increase the probability of pressure injury development.”

Determining all factors that place an individual at risk for a pressure injury is helpful when deciding on appropriate prevention strategies. Once a person is identified to be at risk for a pressure injury, preventive protocols based on the individualized assessment need to be implemented. Recommendations for pressure injury prevention management by level of risk are available on the Braden Web site.[35] However, as the EPUAP/NPIAP/PPPIA has emphasized in the 2019 Clinical Guideline,[1] the individualized risk-based prevention plan of care for any particular patient needs to be based on his or her specific risk factors and needs. The guideline has identified the following risk factor categories: activity/mobility limitations, skin status, perfusion, circulation and oxygenation factors, nutrition indicators, moisture, body temperature, older age, sensory perception limitations, blood markers, and general and mental health status.[1] Specific populations also include individuals in the operating room, critically ill, as well as neonates and children (Table 14-2; Box 14-4).[1]

Table 14-2 Examples of at risk populations

Persons who have/are

- Acutely ill and/or in critical care
- A fractured hip
- Spinal cord injury (SCI)
- Chronic neurological conditions
- Diabetes mellitus
- Older age
- In long-term care or community care
- Experienced trauma and/or prolonged surgery

European Pressure Ulcer Advisory Panel, National Pressure Injury Advisory Panel and Pan Pacific Pressure Injury Alliance. In: E. Haesler, ed. *Prevention and Treatment of Pressure Injuries/Injuries: Clinical Practice Guideline. The International Guideline 2019*. NPUAP/NPIAP/PPPIA, 2019.[1]

BOX 14-4 Risk Factors and Risk Assessment Recommendations and Good Practice statement[1]

EPUAP/NPIAP/PPPIA 2019 Guideline Risk Factors and Risk Assessment Recommendations and Good Practice Statements

2019 Guideline Recommendations

Recommendation Number	Recommendation	Strength of Evidence	Strength of Recommendation
1.1	Consider individuals with limited mobility, limited activity, and a high potential for friction and shear to be at risk of pressure injuries	A	↑↑
1.2	Consider individuals with a category/stage 1 pressure injury to be at risk of developing a category/stage 2 or greater pressure injury	A	↑↑
1.3	Consider the potential impact of an existing pressure injury of any Category/Stage on development of additional pressure injuries	C	↑
1.7	Consider the impact of diabetes mellitus on the risk of pressure injuries	A	↑↑
1.8	Consider the impact of perfusion and circulation deficits on the risk of pressure injuries	B1	↑
1.9	Consider the impact of oxygenation deficits on the risk or pressure injuries	C	↑
1.10	Consider the impact of impaired nutritional status on the risk of pressure injuries	C	↑
1.11	Consider the potential impact of moist skin on the risk of pressure injuries	C	↑
1.12	Consider the impact of increased body temperature on the risk of pressure injuries	B1	↑
1.13	Consider the potential impact of older age on the risk of pressure injuries	C	↑
1.14	Consider the potential impact of impaired sensory perception on the risk of pressure injuries	C	↑
1.15	Consider the potential impact of laboratory blood test results on the risk of pressure injuries	C	↔
1.17	Consider the impact of time spent immobilized before surgery, the duration of surgery, and the American Society of Anesthesiologists (ASA) Physical Status Classification on surgery-related pressure injury risk.	B2	↑
1.19	Consider the impact of skin maturity, perfusion and oxygenation, and presence of a medical device on pressure injury risk in neonates and children	B1	↑↑

Recommendation Number	Recommendation	Strength of Evidence	Strength of Recommendation
1.20	Consider the impact of illness severity and the duration of critical care unit stay on pressure injury risk in neonates and children	B2	↑

Good Practice Statements (GPS)

GPS No.	Statement
1.4	Consider the potential impact of a previous pressure injury on additional pressure injury development.
1.5	Consider the potential impact of alterations to skin status over pressure points on pressure injury risk.
1.6	Consider the potential impact of pain at pressure points on pressure injury risk.
1.16	Consider the potential impact of general and mental health status on pressure injury risk.
1.18	Consider the following as additional risk factors for the development pressure injuries in critically ill individuals: ● Duration of critical care stay ● Mechanical ventilation ● Use of vasopressors ● Acute Physiology and Chronic Health Evaluation (APACHE II) score
1.21	Conduct a pressure injury risk screening as soon as possible after admission to the care service and periodically thereafter to identify individuals at risk of developing pressure injuries.
1.22	Conduct a full pressure injury risk assessment as guided by the screening outcome after admission and after any change in status.
1.23	Develop and implement a risk-based prevention plane for individuals identified as being at risk of developing pressure injuries.
1.24	When conducting a pressure injury risk assessment: ● Use a structured approach ● Include a comprehensive skin assessment ● Supplement use of a risk assessment tool with assessment of additional risk factors ● Interpret the assessment outcomes using clinical judgment.

European Pressure Ulcer Advisory Panel, National Pressure Injury Advisory Panel and Pan Pacific Pressure Injury Alliance. In: E. Haesler, ed. *Prevention and Treatment of Pressure Injuries/Injuries: Clinical Practice Guideline. The International Guideline 2019*. NPUAP/NPIAP/PPPIA, 2019.[1]
See Appendix “Definitions of the EPUAP NPIAP PPPIA 2019 Clinical Guideline Strengths of Evidence and Strengths of Recommendations.”

Questions regarding when to assess patients for pressure injury risk and when to do reassessment have clinical relevance as these aspects of care are very important. The EPUAP/NPIAP/PPPIA clinical practice guideline GPS suggest the initial pressure injury risk assessment to be performed as soon as possible after admission and with any change in clinical status.[1] Reassessment intervals should be based on the acuity of the person. Studies by Bergstrom and Braden[36,37] found that in skilled nursing facilities, 80% of pressure injuries develop within 2 weeks of admission and 96% develop within 3 weeks of admission.

Patient Care to Prevent Pressure Injuries

Preventing pressure injuries can best be accomplished by using a multidisciplinary approach. In the pressure injury prevention protocols and guidelines already mentioned,[1,7–10] a holistic approach to pressure injury prevention is strongly advocated. This includes processes on both the healthcare organizational level and the healthcare provider level that outline an evidence-based plan of care.[1] Healthcare organizations should have a systematic structure for monitoring and managing pressure injury care practices and incidence rates. Quality indicators should be developed and monitored to enable early identification of gaps in practice. These data will support and guide future improvements in care processes needed on the organizational level.[1] The 2019 EPUAP/NPIAP/PPPIA Clinical Guideline provides 20 suggested quality indicators to assist healthcare organizations in implementing the guideline effectively in healthcare settings.

Any good prevention program for the practitioner begins with assessment of the patient's skin. The skin condition should be documented daily in acute- and long-term care settings and at each home care visit. Careful attention to prevention of skin injury during performance of activities of daily living is paramount. The bathing schedule should be individualized[1] based on the patient's age, skin texture, and dryness or excessive oiliness of the skin. Use of nondrying products to clean the skin is recommended. In a study by Carr and Benoit, the incidence of pressure injuries decreased from 7.14% at baseline to 0% at the end of the study through education of nonlicensed staff about the use of protective skin barriers and implementation of a comprehensive interventional patient hygiene management program focused on bathing and incontinence care.[38] The aim should always be to keep the skin clean and well hydrated with cleansing after each incontinence episode. Avoiding the use of alkaline soaps and cleansers as well as avoiding vigorous skin scrubbing and rubbing of vulnerable skin at risk for breakdown are key to skin protection regimens. Daily baths may be replaced with "lotion"

baths in vulnerable populations to prevent skin damage associated with lifting and handling.

For the incontinent patient, moisture barrier products such as acrylate creams or water-repellant ointments should be considered as treatment options. Soiled skin should be cleaned immediately and products to protect the skin applied once the area is well dried off. If fecal containment products or devices are used, it is important to follow the correct methods of application to prevent a potential device-related injury severely aggravating already vulnerable skin.[39,40] The underlying causes for incontinence should always be determined with appropriate measures implemented to either control or manage the condition.

Vulnerable skin should be additionally protected from injury. Citing a B1 strength of evidence, the EPUAP/NPIAP/PPPIA recommends prophylactic use of soft silicone multilayered foam dressings applied on the heel, sacrum, or other at-risk locations to prevent pressure injuries.[1,41,42] In a study of 93 high-risk patients in a surgical trauma intensive care unit, the use of intervention bundles that included a prophylactic soft silicone dressing product resulted in zero pressure injuries.[33] In addition, five-layer silicone border foam dressings applied prophylactically to the sacrum and heels have been recommended to minimize the risk for pressure injuries.[39,40] A series of published large randomized controlled trials called the "BORDER" trials have demonstrated the clinical efficacy of the above dressings in prevention of sacral and heel pressure injuries in acute care settings[43,44]. These dressings reduce the body–bed friction and absorb shear internally through intradressing deformations, which then lessen the mechanical loading transferred to the soft tissues in vicinity to the bony prominences under the dressing.[39–50]

Although a review of the literature suggests that one type of massage may be beneficial for at-risk patients,[46,48] most clinical guidelines recommend against massaging reddened bony prominences because this can lead to further tissue damage.[1,8] It is important to keep the patient's heels slightly elevated and not resting directly on the surface off the bed.[43,44] The use of pillows, wedges, and foot elevation devices is indicated as preventative measures (see Box 14-3). By flexing the knee when elevating the leg, pressure to the

Achilles tendon and impaired venous return is prevented.[1] Best practices for preventing heel pressure injuries in the orthopedic population exist and may provide valuable guidance to healthcare providers in proactive protective management of vulnerable heels. [51–60]

The 2019 EPUAP/NPIAP/PPPIA Clinical Guidelines suggest "conducting a comprehensive skin and tissue assessment for all individuals at risk of pressure injuries."[1] This includes a head-to-toe examination for signs of erythema, edema, maceration, temperature changes, moisture presence or damage, pain at pressure points, and change in tissue consistency and tensile strength.[1] Consider using a subepidermal moisture/edema measurement device as an adjunct to skin assessment. Skill in the evaluation of skin tone is an important especially in assessment of darkly pigmented skin.[1]

Practice Point

Keep the patient's heels off the mattress!

Be careful not to drag your patient during transfers or position changes. Use appropriate devices, such as sliding/turning sheets or mechanical lifters, to prevent friction injuries to skin and shear injuries to deeper tissues of patients. Consider using some of the new textiles on the market to aid in transfers and assisting to protect skin in at-risk individuals. Use the 30-degree lateral position for patients in bed to help on easier turning procedures. Keep the head of the bed as flat as possible as shear forces increase substantially above 30 degrees to prevent shearing injuries, unless contraindicated due to the clinical condition of the patient.[54,55]

The role of nutrition in pressure injury interventions has been controversial. In a large prospective cohort study, Bergstrom and Braden[36] found that nursing home residents who developed pressure

injuries had a significantly lower intake of protein. Clinicians need to ensure that a patient's caloric, protein, vitamin, and mineral needs are met. The 2019 EPUAP/NPIAP/PPPIA Clinical Guideline recommends providing patients who are at risk of malnutrition or stage 2 or greater pressure injuries, with a high-caloric, high-protein arginine, zinc, and antioxidant oral nutritional supplement or enteral formula.[1,61]

Physical and occupational therapists are important members in the pressure injury team and a valuable resource for maximizing patient mobility. Their expertise in selecting appropriate sized wheelchairs and ability to conduct seating angle and postural alignment evaluations can't be overemphasized. Patients who are confined to a chair should be repositioned every hour, with small shifts in weight made every 15 minutes. In the past, many clinicians considered turning and repositioning bedridden patients every 2 hours to be a standard of care. However, the EPUAP/NPIAP/PPPIA Clinical Guideline recommends that repositioning should take into account the individual's condition and type of pressure redistribution support surface the patient is positioned on.[1] The most appropriate turning interval applicable for all patients has yet to be determined by research. A 2-hour interval may be too long for some patients, whereas for others, every 2 hours may not be practical such as for palliative care patients where frequent repositioning cause more pain and suffering, than benefit. Because there is no standard time interval or frequency for repositioning all patients, a repositioning schedule needs to be individualized for each patient. The most recent EPUAP/NPIAP/PPPIA Clinical Guideline[1] provides evidence that repositioning frequency may be influenced by the type of support surface being used. "Use a high specification reactive single layer foam mattress or overlay in preference to a foam mattress without high specification qualities for individuals at risk of developing pressure injuries."[1] Bergstrom and colleagues found that for moderate- to high-risk residents in long-term care who were on high-density foam mattresses, there were no differences in pressure injury development with turning schedules of every 2, 3, or 4 hours.[62]

Practice Point

Repositioning schedules should take into consideration the condition of the patient and the support surface in use.[1]

The EPUAP/NPIAP/PPPIA Clinical Guideline has provided new recommendations regarding the repositioning of critically ill persons. This includes the following GPS from the guideline: Start a repositioning schedule as soon as possible after the patient is admitted and revising the schedule for patient repositioning based on the patient's skin and tissue tolerance, general medical condition, overall treatment objectives, patient comfort and pain levels.[1] They recommend considering the use of slow, gradual turns that allow the patients sufficient time to stabilize their hemodynamic and oxygenation status.[1] Also, clinicians should consider more frequent small shifts for critically ill persons that cannot tolerate frequent major shifts in position.[1] If it is determined for a medical reason that a patient who is hemodynamically unstable, or has spinal instability, cannot be turned, clinicians should discuss options with the physician in determining a workable patient care plan.

Rastinehad reported that the use of appropriate support surfaces decreases the incidence of pressure injuries in at-risk oncology patients.[63] Devices such as donut-shaped positioners should not be used as they concentrate the transfer of bodyweight forces through narrow support regions, thereby creating elevated tissue stresses, particularly in shear, which endangers tissue integrity and viability at these peak loading sites. The type and number of layers used underneath a patient may make a different in pressure injury incidence.[1] Limit the amount of linen and incontinence pads placed on the support surface/bed.[1] The general rule of thumb is that less is best. Evidence suggests that silk like bed linens or cotton-blend fabrics reduce shear, friction, and pressure injury incidence.[64–66] The results

of a Canadian study have led to a model for use in support surface selection.[67] See the NPIAP Web site (http://www.npiap.com) to review the latest definitions of physical concepts related to support surfaces as developed by the NPUIP Support Surface Standards Initiative.[68] (Also, see Chapter 11, Pressure Redistribution: Seating, Positioning, and Support Surfaces.)

Ongoing monitoring and documentation of skin condition are essential. In a pilot study, Horn et al.[69] found that establishing a multidisciplinary team and redesigning documentation processes for certified nursing assistants in several long-term care facilities throughout the United States resulted in a decrease of the required facility documentation. For the seven facilities included in the study, there was a combined 33% reduction in pressure injuries.[69]

Milne et al.[70] decreased facility-acquired pressure injury prevalence from 41% to 4.2% in a 108-bed long-term acute care hospital (LTACH) by developing policies that were supported by published clinical practice guidelines and incorporating them into the facility's plan of care. They established a wound care team, improved documentation methods, and conducted educational interventions for staff. They also reviewed the facility's wound products and revised its electronic record. All of these efforts resulted in care improvement outcome.[70]

Pressure Injuries Staging

A comprehensive wound assessment includes many parameters, one of which is staging. (See Chapter 6, Wound assessment.) Once the wound etiology is known, the correct classification system to describe the wound can be selected. For example, arterial and venous ulcers are described by their characteristics. Diabetic or neuropathic ulcers can be classified by using the American Diabetes Association system, Wagner Grading System for Vascular Wounds, or San Antonio Diabetic Wound Classification System. DO NOT USE THE PRESSURE INJURY CLASSICATION DEFINITIONS (STAGING OR CATEGORIES) to describe arterial, venous, diabetic or neuropathic ulcer; skin tears; or moisture-associated skin damage (MASD). The pressure injury staging system of the NPIAP is designed specifically for pressure injuries. An outcome of the first NPIAP consensus conference[71] was the NPIAP staging system for pressure injuries, based on staging systems by Shea[72] and the International Association of Enterostomal Therapists[73] (now called WOCN). In February 2007, NPIAP once again revised its staging system and categories[74] The latest NPIAP revision of the pressure injury classification system included six categories or stages in the 2019 EPUAP/NPIAP/PPPIA international pressure injury guideline along with other classification systems used around the world.[1]

Pressure injury staging is a classification system that describes the level of tissue destroyed. It provides practitioners with a common language to communicate the clinical condition of pressure injuries effectively. This staging system should only be used to describe pressure injuries and not for other types of skin or wound injuries.[75] NPIAP states that mucous membrane pressure injuries should not be staged using its pressure injury staging system.[25] Mucosal pressure injuries (MPrI) are “pressure injuries found on mucous membranes with a history of a medical device in use at the location of the injury”[25] (see Fig. 14-5). Staging is only part of the total assessment of an injury; a comprehensive assessment also includes factors such as the state of surrounding skin and presence of infection, among others. (See Chapter 6, Wound assessment.)

After the present on admission (POA) indicator went into effect in US acute care settings, confusion existed as to whether nurses could continue to stage pressure injuries. An NPIAP position statement[75] as well as information in the literature from the American Nurses Association (ANA) reaffirm that pressure injury staging is within the scope of practice for registered nurses (RNs).[76,77]

NPIAP Classification of Pressure Injuries: Staging Definitions

Pressure injuries are classified according to the amount of visible tissue loss.[1] Necrotic pressure injuries that covers all of the pressure injury cannot be staged numerically because visualization of the wound bed is necessary to determine the level of tissue involvement; therefore, necrotic pressure injuries that obscure the base of the wound are classified as unstageable pressure injuries. Numerical staging of necrotic pressure injuries should be done after the necrotic tissue is removed. (See Chapter 8, Wound débridement.)

The 2019 International EPUAP/NPIAP/PPPIA guideline describes a variety of classification systems commonly used in the United States, Europe, and the Pan Pacific region.[1] This chapter will only be addressing the NPIAP classification system, which is commonly used in the United States. The classification system for the American Centers for Medicare and Medicaid Services (CMS) in post–acute care uses CMS adapted definitions that are required to be used in post–acute care settings such as long-term care (LTC) and long-term acute care hospitals (LTCH).

"Use a pressure injury classification system to classify and document the level of tissue loss" is a good practice statement.[1]

The EPUAP/NPIAP/PPPIA classification system[1] for staging of pressure injuries is described below.

Stage 1

The original definition of a stage 1 pressure injury was "nonblanchable erythema of intact skin, the heralding lesion of skin ulceration."[71] Given the diversity of people with different skin pigmentation, detecting stage 1 pressure injuries can be a challenge if clinicians only use color as an indicator. In 1997, to provide a more culturally sensitive definition, NPIAP revised the definition of a stage 1 injury to include indicators that went beyond color. NPIAP continued to refine this definition to include "intact skin with nonblanchable redness of a localized area

usually over a bony prominence." In darkly pigmented skin, the area may not have visible blanching and its color may differ from surrounding skin (see Box 14-1).

Persons with darkly pigmented skin have the lowest prevalence of stage 1 pressure injuries.[78,79] The incidence of pressure injuries was higher in people with darkly pigmented skin in several studies conducted by Lyder and colleagues.[80–84] Sprigle and colleagues[84] found that local warmth or coolness was present in 85% of patients with stage I pressure injuries. Rosen and colleagues[85] used a quality improvement program for educating staff on assessing the subtle differences in skin color, texture, and warmth in individuals with darkly pigmented skin. This resulted in eliminating the racial disparity differences in PI rates between darkly pigmented and lightly pigmented residents.[86] Differences in PI incidence rates may not be due to skin tone differences, but rather, further research on cultural, social, or economic factors may shed new light on this viewpoint.[87–97]

Stage 2

Stage 2 pressure injuries (PI) have partial-thickness tissue loss where the dermis is exposed and visible.[1] (See Box 14-1 for additional definitions.) These pressure injuries are often described as "shallow", but this is not an accurate descriptor and can be misleading. A stage 2 pressure injury does not extend into deeper tissue such as muscle, bone or subcutaneous tissue. A stage 2 PI can also present as a serum-filled blister. Differential diagnosis is important as sometimes a stage 2 pressure injury is misidentified as a skin tear, MASD, MARSI, or burn. Getting the etiology (cause) correct is important—if it's pressure then it is a pressure injury and the NPIAP classification system can be used.

Stages 3 and 4

Stage 3 and stage 4 pressure injuries are considered to have full-thickness skin loss.[1] (See Box 14-1 for additional definitions.) In pressure injuries at both these stages, undermining and tunneling may be present. A major difference between stage 3 and stage 4 is that in a

stage 3 PI, fascia, muscle, tendon, ligament, and bone are not exposed.[1]

Deep Tissue Pressure Injury

Clinicians often struggle with the concept of DTPI[96,97] that presents as a deep red, purple, or maroon color, seen in the heel area. Typically, these wounds appear as dusky, boggy, or discolored purple or maroon areas (Fig. 14-7). Sometimes, they appear a few days after surgery as a discolored area on the sacrum and may be misidentified as a burn.[98] These areas may deteriorate rapidly from intact skin to deep open wounds.

Figure 14-7. Deep tissue injury. © J. M. Levine, MD

What is DTPI?[19,90] Can these pressure injuries be prevented and, if so, how? What is the best treatment? Clinicians have sought guidance about this particular form of pressure injury for a long time. Black reports the finding of Baharestani that 36% of their DTPI cases resolved and 90% of these patients had anemia.[99] Initially, DTPI lesions have the appearance of a deep bruise and may herald the subsequent development of a stage 3 or 4 pressure injury even with optimal management. Honaker, Brockopp, and Moe have described precipitating events to DTPI, as well as a research tool, which evaluates the severity of deep tissue injury.[100,101]

Early Detection of DTPI

The pressure injury staging system relies on visual inspection of the skin. Newer technologies may hold the promise of early detection for pressure injury *before* visible signs of tissue destruction can be seen. One study reported on the preulcerative changes in long-term residents using diagnostic ultrasound.[102] Koerner et al. used thermal imaging to assess for DTPI for patients on admission in an ICU, which resulted in early detection of DTPI and implementation of new wound prevention protocols.[103] Another new FDA-authorized and commercially available technology for early detection of DTPIs includes a handheld noninvasive subepidermal moisture (SEM) scanner, which records biocapacitance differences around the sacrum and posterior heels of supine patients. More studies on this will follow in future.

Unstageable Pressure Injury

An unstageable pressure injury is a PI where the wound bed is covered with necrotic tissue such as eschar or slough tissue. (See Box 14-1 for additional definitions.)[1] The PI wound bed is no longer visible to see the level of tissue involvement to determine which numerical staging number to assign to the pressure injury. If débridement is appropriate with the patient care goals and vascular supply condition, then once the pressure injury wound can be seen, a numerical pressure injury stage can be used.

Practice Point

Unstageable pressure injuries are those covered with necrotic (slough and eschar) tissue. Until the necrotic tissue is removed, the injury can't be visualized accurately and is therefore unstageable in patients in all care settings.

Staging Concepts

Reverse/back staging

Do not reverse or "back stage" a pressure injury as it heals.[1] When a pressure injury is healing, the wound does not go from a higher numerical stage pressure injury to a lower numerical stage pressure injury. For example, do not incorrectly classify a healing stage 4 pressure injury as going from a stage 4 to a stage 3, to a stage 2 and to a stage 1 as it heals.

Staging Competency

Accuracy in staging pressure injuries is a challenge. Nurses have reported being less confident in identifying stage 3 injuries.[92–96]

Data from a 2019 nursing survey revealed that just about one-fourth of the nurses correctly responded that a mucosal membrane pressure injury cannot be staged, with 27.97% incorrectly believing it should be staged and over half of the nurses (58%) answering that they did not know whether mucosal membrane pressure injury should be staged[93] (they should not according to the NPIAP).[1] This is an important opportunity for additional education needed for nurses as most have incorrect knowledge. Based on the NPIAP recommendations, Delmore and Ayello have provided an enabler to remind nurses that mucosal pressure injuries should be counted but

cannot be staged due to the fact that mucosa does not keratinize and therefore cannot be staged.[93]

Clinicians can test their ability to classify pressure injuries by taking the ePUCLAS$_4$ staging test (available in English and other languages) on the EPUAP Web site (http://www.epuap.org)[94] or the quiz on the National Database of Nursing Quality Indicators (NDNQI) educational modules.[95]

Treatment of Pressure Injuries

Avoidable Pressure Injury

NPIAP issued a definition of unavoidable pressure injuries that was applicable across all care settings.[104] It is based on the CMS definition of "unavoidable" from Long-Term Care F-Tag 314.

Practice Point

"Unavoidable PI may occur even though providers have evaluated the individual's clinical condition and PI risk factors have been evaluated and defined and interventions have been implemented that are consistent with individuals' needs, goals, and recognized standards of practice. Further, an unavoidable PI was characterized as one that occurs even though providers have monitored and evaluated the impact of preventive interventions and revised these approaches as appropriate."[104]

Although consensus was reached on 15 items at the 2014 NPIAP conference,[104] the debate continues as to whether all pressure injuries are avoidable.[104–106] Several guidelines are now available for clinicians to use in planning treatment and setting achievable skin- and wound-related objectives.[1,5–10] Clinical guidelines provide the foundation for establishing evidence-based pressure injury management.

Treatment

The wound bed preparation model has been applied to pressure injury prevention and treatment very effectively (Fig. 14-8).[107] A pressure injury won't heal unless the underlying causes are effectively managed. A general assessment should include identifying and effectively managing the patient's medical diseases, health problems

(such as urinary incontinence), nutritional status, individual concerns (such as pain level),[108] and psychosocial health. Unless these major areas are effectively addressed, the probability of the pressure injury achieving healing is unlikely.

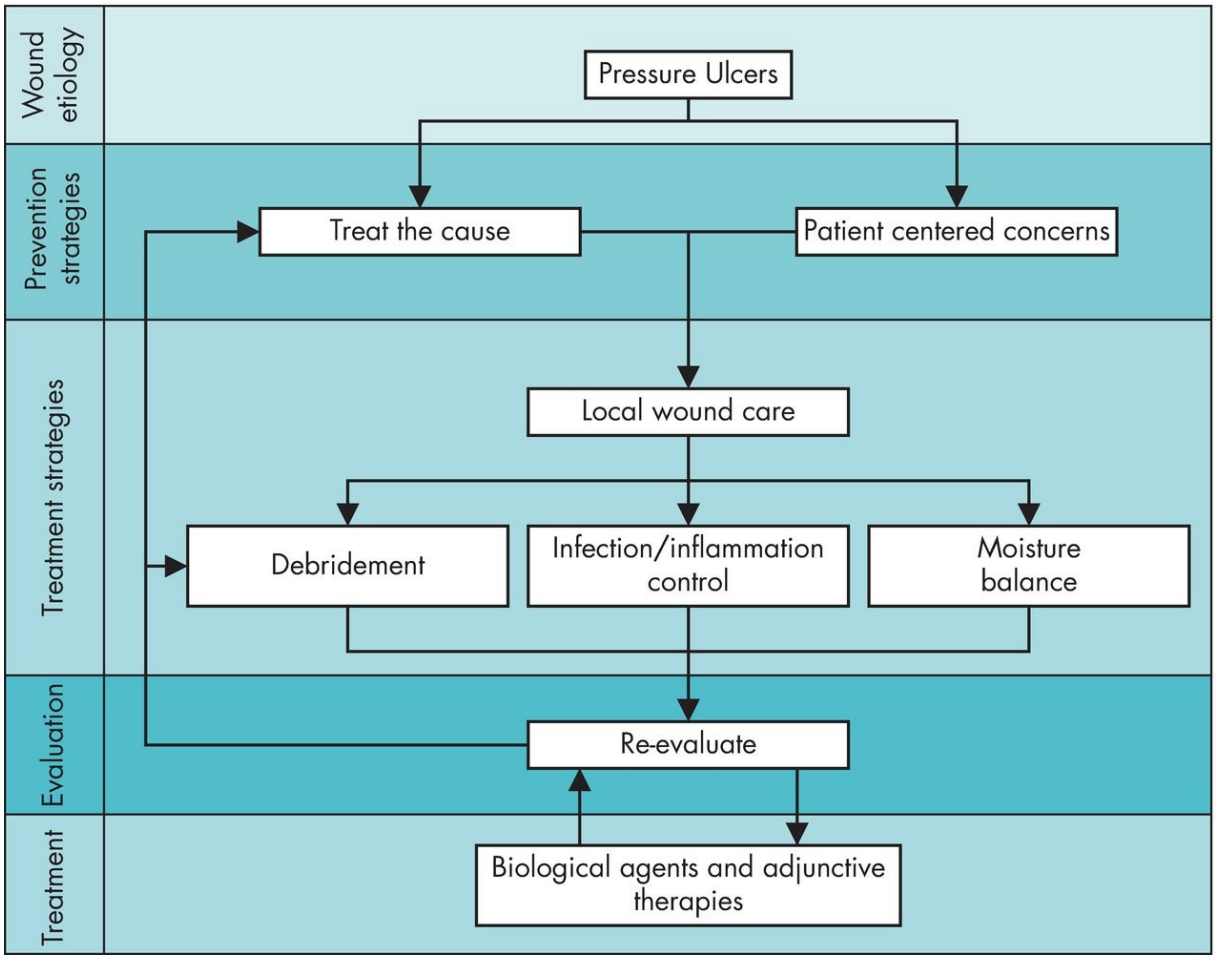


Figure 14-8. Pressure ulcer treatment and prevention using the wound bed preparation model. (Used with permission, Dr. David Keast.)

The comprehensive local management of pressure injuries includes cleaning, controlling infections, débridement, dressings that promote a moist wound environment (if a healable wound), nutritional support, and redistribution of pressure (repositioning and use of support surfaces). (See Chapter 11, Pressure Redistribution: Seating,

Positioning, and Support Surfaces.) The use of adjunctive therapies to heal pressure injuries should be considered for recalcitrant pressure injuries. Options are discussed later in the chapter.

Monitoring Healing

Because reverse staging of pressure injuries to monitor healing is inappropriate, several instruments have been developed and validated to assess the healing of pressure injuries.

Tools That Measure Healing

The two most widely used tools to measure healing in pressure injuries are the Bates-Jensen Wound Assessment Tool (BWAT)[109] and the Pressure Ulcer Scale for Healing (PUSH).[110] The BWAT is composed of 13 variables that provide a numerical indicator of the status of the pressure injury (healing or deteriorating).[111] The score ranges from 1, which indicates tissue health (or healed), to 65, which indicates wound degeneration. The variables that compose the BWAT score include wound size (length and width), depth, edges, undermining, necrotic tissue type, necrotic tissue amount, exudate type, exudate amount, skin color of surrounding wound, peripheral tissue edema, peripheral tissue induration, granulation tissue, and epithelialization. The BWAT provides a comprehensive assessment of pressure injuries[109] and is currently being evaluated for use with other wound types.[112]

The PUSH tool[110,112] uses only three variables—surface area (length and width), exudate amount, and tissue appearance—to derive a numerical indicator of the status of the pressure injury (Box 14-5). A score of 0 indicates that the pressure ulcer has healed, while the highest score of 17 indicates wound degeneration. The PUSH tool intentionally takes a “minimalist approach.” Using research databases in its development, the PUSH tool seeks to select the minimum number of assessment parameters needed to monitor ulcer healing or deterioration. Its brevity and accuracy in monitoring make the PUSH tool ideal for quality assurance monitoring of large groups of patients and identifying patients who are deteriorating and require reassessment and possibly treatment changes. The PUSH tool isn’t intended to provide a comprehensive assessment of pressure injuries. It has been used in numerous research studies and has been integrated into several computer-based wound assessment

systems.[113] The NPIAP has plans to revise the PUSH tool in the near future.

A survey of 103 respondents found the PUSH tool reliable and easy to use.[114]

BOX 14-5 NPUAP PUSH Tool

PATIENT NAME:				PATIENT ID. No.			
ULCER LOCATION:				DATE:			

Directions

Observe and measure the pressure ulcer. Categorize the ulcer with respect to surface area, exudate, and type of wound tissue. Record a subscore for each of the ulcer characteristics. Add the subscores to obtain the total score. A comparison of total scores measured over time provides an indication of the improvement or deterioration in pressure ulcer healing.

LENGTH	**0** 0 cm^2	**1** <3 cm^2	**2** 0.3–0.6 cm^2	**3** 0.7–1.0 cm^2	**4** 1.1–2.0 cm^2	**5** 2.1–3.0 cm^2	**Blank**
× **WIDTH**		**6** 3.1–4.0 cm^2	**7** 4.1–8.0 cm^2	**8** 8.1–12.0 cm^2	**9** 12.1–24.0 cm^2	**10** >24.0 cm^2	**Subscore**
EXUDATE AMOUNT	**0** None	**1** Light	**2** Moderate	**3** Heavy		**Subscore**	
TISSUE TYPE	0 Closed	**1** Epithelial tissue	**2** Granulation tissue	**3** Slough	**4** Necrotic tissue		**Subscore**
							Total score

LENGTH × WIDTH

Measure the greatest length (head to toe) and the greatest width (side to side) using a centimeter ruler. Multiply these two measurements (length × width) to obtain an estimate of surface area in square centimeters (cm^2). Do not guess! Always use a centimeter ruler and always use the same method each time the ulcer is measured.

EXUDATE AMOUNT

Estimate the amount of exudate (drainage) present after removal of the dressing and before applying any topical agent to the ulcer. Estimate the exudate as none, light, moderate, or heavy.

TISSUE TYPE

This refers to the types of tissue that are present in the wound (ulcer) bed. Score as a "4" if any necrotic tissue is present. Score as a "3" if any amount of slough is present and necrotic tissue is absent. Score as a "2" if the wound is clean and contains granulation tissue. Score as a "1" if the wound is superficial and reepithelializing. Score as a "0" if the wound is closed.

4–Necrotic tissue (eschar): Black, brown, or tan tissue that adheres firmly to the wound bed or ulcer edges and may be either firmer or softer than surrounding tissue.

3–Slough: Yellow or white tissue that adheres to the ulcer bed in strings or thick clumps or is mucinous.

2–Granulation tissue: Pink or beefy-red tissue with a shiny, moist, granular appearance.

1–Epithelial tissue: For superficial ulcers, new pink or shiny tissue (skin) that grows in from the edges or as islands on the ulcer surface.

0–Closed/resurfaced: The wound is completely covered with epithelium (new skin).

PUSH Tool version 3.0, ©1998 National Pressure Ulcer Advisory Panel. Used with permission.

Although tools exist for measuring pressure injury healing, more evidence is needed to determine pressure injury healing rates.[115–118] Among chronic wounds, pressure injuries have the slowest healing rate—0.077 mm/d, with 2.156 mm of healing expected at 4 weeks.[117] The use of high-frequency portable ultrasound to measure wound healing has been introduced. The use of this technology, which can capture three-dimensional measurements, has been shown to be quite beneficial in objectively monitoring healing. Ultrasound is also “color blind”; that is, it can detect stage 1 pressure ulcers in darkly pigmented skin.[119]

Principles of Local Wound Care

After addressing the cause and patient-centered concerns, healing a pressure ulcer using the wound bed preparation model requires attention to determining the wound prognosis (healable, maintenance, or nonhealable wound)[120] and applying the principles of local wound care (débridement, infection management, moisture balance before attention to the edge).

Cleansing

Cleaning the pressure injury to remove devitalized tissue and decrease bacterial burden is often recommended. EPUAP/NPIAP PPPIA also recommends cleansing the surrounding skin.[1] Pressure ulcers can exhibit delayed healing in the presence of high levels of bacteria.[121] Solutions that don't traumatize the healing injury should be used.[1,122,123] Normal saline solution (0.9%) is usually recommended because it isn't cytotoxic to healthy tissue.[1] Although the active ingredients in newer wound cleaners may be noncytotoxic (surfactants), the inert carrier may be cytotoxic to healthy granulation tissue. Review of all ingredients is warranted. Hellewell et al.[124] found that antiseptic cleaners were the most cytotoxic to granulation tissue. An in vitro study found relative toxicity indexes ranging from 0 to 100,000 with saline and Shur-Clens to be least toxic to fibroblasts, while Dial antibacterial soap and Ivory liquid gel were the most toxic.[121] Least toxic to keratinocytes were Biolex, Shur-Clens, and Techni-Care, while hydrogen peroxide, modified Dakin's solution, and povidone (10%) were most toxic.[125] The 2019 guideline provides a table that summaries topical antimicrobial therapies with polyhexamethylene biguanide (PHMB) having low to no cytotoxicity and octenidine dihydrochloride (OCT) showing high from in vitro tests.[1] Sterile normal saline and sterile water have no cytotoxicity.[1]

The mechanical method used to deliver the cleaning agent must provide enough pressure to remove debris without presenting trauma to the ulcer bed.[1] Optimal pressure to clean a pressure injury is between 4 and 15 pounds per square inch (psi).[126] A 35-mL syringe

with a 19-gauge needle creates an 8-psi irrigation pressure stream,[1] which was found to be more effective in removing bacteria than other irrigation pressures.[127] It should be noted that irrigation pressures exceeding 15 psi can cause trauma to the ulcer bed and may drive bacteria into the tissue.[128] New technology such as battery-powered, disposable irrigation devices can provide an alternative to the syringe and needle system to loosen wound debris.

Débridement

The presence of necrotic devitalized tissue promotes the growth of pathological organisms and prevents wounds from healing.[129] Débridement is, therefore, a very important step in the local management of pressure injuries. There's no optimal débridement method; selection should be based on the goals of the patient, absence or presence of infection, amount of necrotic tissue present, and economic considerations for the patient and the facility.

Many types of débridement, including surgical, autolytic, enzymatic, mechanical, biological, and laser, are available. (See Chapter 8, Wound débridement.) However, surgical (or sharp laser) débridement is considered by many to be the most effective form of débridement because it involves the cutting away (with a scalpel) of necrotic tissue.[130] In addition, surgical débridement is relatively quick and can be done at the bedside. Surgical débridement is essential when cellulitis or sepsis is suspected.[131] Autolytic débridement involves the use of a semiocclusive or occlusive dressing (e.g., hydrocolloids, hydrogels) and employs the body's own natural enzymes to digest necrotic tissue. Autolytic débridement takes much longer than does sharp débridement. Watch closely for signs and symptoms of infection. Enzymatic débridement uses proteolytic enzymes to remove necrotic tissue. In the United States, papain urea, and trypsin are no longer available for use; only collagenase can be used for enzymatic debridement. Mechanical débridement uses wet-to-dry gauze to adhere to the necrotic tissue, which is then removed. Upon removal of the gauze dressing, necrotic tissue and wound debris are removed. However, healthy granulation tissue is also removed, which causes microtrauma to the wound bed and can delay wound healing.[130]

Pressure Redistribution

The use of support surfaces is an important consideration in redistributing pressure. (See Chapter 11, Pressure Redistribution: Seating, Positioning, and Support Surfaces.) The NPIAP Support Surface Standards Initiative redefined the physical concepts related to support surfaces.[68]

Few studies demonstrate significant differences within the support surface classifications and preventing or healing pressure ulcers. Therefore, the level and type of risk factors should guide the level and type of support surface selected. The EPUAP/NPIAP/PPPIA clinical practice guideline recommends that a patient should not be positioned directly on a pressure injury unless there are pressure injuries on multiple anatomical sites.[1]

Dressings

The use of moist wound therapy dressings is a major component in managing a healable pressure ulcer. (See Chapter 9, Wound Treatment Options.) At present there is no shortage of dressing materials available with a myriad of options that is specifically designed for pressure injury management. Dressings can be broken down into several classifications: gauze, nonadherent gauze, transparent films, hydrocolloids, foams, alginates, hydrogels, collagens, antimicrobials, composites, and combinations. Matching the dressing to the wound bed characteristics is essential. A guiding principle is to maintain a moist environment for a healable wound.

Although nongauze dressings are usually more expensive than gauze dressing, less frequent dressing changes, faster healing rates, and decreased rates of infection can make non–gauze-based dressing more cost-effective over time.[132–135] It's also important to note that wet-to-dry gauze dressings are a form of mechanical débridement and shouldn't be used on injuries with good granulation tissue. Use moist gauze dressings to maintain an appropriately moist wound environment when advanced wound dressings are not an option.[1] No specific dressing heals all pressure injuries within an ulcer classification. Consequently, a careful assessment of the pressure injury, the patient's needs, and environmental factors (frequency of

dressing changes to increase adherence) must be considered. (See Chapter 9, Wound Treatment Options.)

Nutrition

Nutrition is important to maintain the body in positive nitrogen balance,[1] thereby increasing wound healing. Patients should be assessed and screened for nutritional status, including weight, weight history (e.g., significant weight loss), and adequacy of nutrient and fluid intake.[1] It's important to provide calories (30 to 35 calories/kg body weight) and protein for malnourished patients with pressure injuries.[1] Always check renal status to make sure that the patient's kidneys can handle the protein load.[1] Initiating nutritional supplements or enteral formula for adults with stage 2 or greater pressure injuries who are malnourished or at risk of malnutrition is a key intervention.[1] The EPUAP/NPIAP/PPPIA clinical guideline recommends[1] that the benefits and harms of enteral and parental nutritional support be discussed in light of patient goals and preferences when the patient is unable to meet nutritional needs through oral intake.[1] There is new evidence supporting high- calorie, high-protein arginine, zinc, and antioxidant supplements or enteral feedings to support pressure injury healing in malnourished patients.[1]

Control of Infections

All pressure injuries will become colonized with both aerobic and anaerobic bacteria; therefore, pressure injuries aren't sterile wounds (See Chapter 7, Wound, Bioburden, and Infection.) (Box 14-6).

BOX 14-6 Infection and Biofilms

Recommendation

Recommendation No.	Recommendation	Strength of Evidence	Strength of Recommendation
13.1	Have a high index of suspicion of local infection in a pressure injury in the presence of: ● Delayed healing ● Lack of signs of healing in the preceding two weeks despite appropriate treatment ● Larger size and/or depth ● Wound breakdown/dehiscence ● Necrotic tissue ● Friable granulation tissue ● Pocketing or bridging in the wound bed ● Increased exudate, or change in the nature of the exudate ● Increased warmth in the surrounding tissue ● Increased pain ● Malodor	B1	↔

Good Practice Statement (GPS)

GPS No. 13.2	Have a high index of suspicion of biofilm in a pressure injury in the presence of: ● Failure to heal despite appropriate antibiotic therapy ● Recalcitrance to appropriate antimicrobial therapy ● Delayed healing despite optimal treatment ● Increased exudate ● Increased poor granulation or friable hypergranulation ● Low-level erythema and/or low-level chronic inflammation ● Secondary signs of infection

European Pressure Ulcer Advisory Panel, National Pressure Injury Advisory Panel and Pan Pacific Pressure Injury Alliance. In: E. Haesler, ed. *Prevention and Treatment of Pressure Injuries/Injuries: Clinical Practice Guideline. The International Guideline 2019*. NPUAP/NPIAP/PPPIA, 2019.[1]

Clean technique is customarily used when treating pressure injuries. If a pressure injury may be infected (independent of a puncture biopsy), most experts assess for the amount of drainage and odor and examine the surrounding tissue for cellulitis. Tissue biopsy or semiquantitative swab techniques are the two preferred methods.[1] Wound bed cultures, though, should serve the purpose to guide systemic antibiotic treatment rather than be a tool to diagnosis infection. It should be noted that some infected pressure injuries may not demonstrate the

typical signs and symptoms associated with infection; rather, they may appear as nonhealing wounds.

A pressure injury will not heal until infection is controlled. Nonhealable or infected wounds may benefit from short-term use of 1% povidone–iodine (antiseptic). “Use topical antiseptics in tissue-appropriate strength to control microbial bioburden and to promote healing in pressure injuries that have delayed healing.”[1] Clearly, additional research is needed to examine the role of topical antiseptics & antibiotics in decreasing bacterial loads in pressure injuries. Systemic antibiotics should only be used when a systemic infection is suspected.” Use systemic antibiotics to control and eradicate infection in individuals with pressure injuries and clinical evidence of systemic infection.”[1] The 2019 EPUAP/NPIAP/PPPIA Clinical Guideline also has a C level recommendation that states to “use topical antiseptics that are active against biofilm in tissue-appropriate strengths in conjunction with regular débridement to control and eradicate suspected (or confirmed) biofilm in pressure injuries with delayed healing.”[1] When exposed bone is present in a pressure injury, evaluate for osteomyelitis when healing has not occurred.[1]

Biophysical Agents/Adjunctive Therapies

The use of adjunctive therapies is the fastest-growing area in pressure ulcer management. Adjunctive therapies include electrical stimulation, hyperbaric oxygen therapy, radiant heat, growth factors, and skin equivalents among others.

The EPUAP/NPIAP/PPPIA clinical practice guideline recommendation on pulsed current electrical stimulation is at the A level of evidence.[1] Electrical stimulation is the use of electrical current to stimulate a number of cellular processes, such as increasing fibroblasts, neutrophils, macrophages, collagen synthesis, and DNA synthesis and increasing the number of receptor sites for specific growth factors.[136–138] Electrical stimulation appears to be most effective on stage 2, 3, and 4 pressure injuries[1] that are unresponsive to traditional methods of healing. Although there are much data to suggest that electrical stimulation is effective in healing pressure injuries, the optimal electrical charge needed to stimulate pressure

injury healing remains unclear. The literature suggests that an optimal electrical charge of 300 to 500 μA/second produces positive effects on the pressure ulcer.[139] However, additional research is needed to determine the optimal electrical charge based on the characteristics of pressure injuries (e.g., stage, depth, and amount of drainage). There is evidence supporting the use of noncontact low-frequency ultrasound as an adjunct to treating stage 3, stage 4, and DTPI; high-frequency ultrasound at 1 MHz as an adjunct to treating stage 3 and stage 4 pressure injuries; and continued support for negative pressure wound therapy.

EPUAP/NPIAP/PPPIA 2019 guidelines concluded that there was insufficient evidence to recommend hyperbaric or topical oxygen therapy for the treatment of pressure injuries.[1]

Growth factors and skin equivalents are emerging methods of healing pressure injuries. There is evidence supporting the use of platelet-rich plasma, and platelet-derived growth factors as well as collagen dressings for promoting healing in stages 3 and 4 pressure injuries.[1] One multicenter, randomized, double-blind study examining the use of rhPDGF-BB exists in literature.[140] This study enrolled 45 patients with stage 3 or 4 pressure ulcers who were randomized to treatment group 1 (300 μg/mL of rhPDGF), treatment group 2 (100 μg/mL rhPDGF), or treatment group 3 (placebo). After 4 weeks of treatment, patients in group 1 had a 40% reduction in ulcer area, group 2 had a 71% reduction in ulcer area, and group 3 had a 17% reduction in pressure ulcer area.[140] From these results, it is clear that the use of growth factors may have a crucial impact on the future of wound healing. However, additional research is needed to evaluate the efficacy of specific growth factors in healing pressure injuries.

Prevalence and Incidence

It has been said, “What can be measured can be managed.” Yet research has shown that just a less than half of the nurses who responded to a survey (42.27%) knew their unit’s pressure injury incidence rate and just a little more than one-third (37.50%) of nurses know their facilities prevalence rate.[91]

To improve pressure injury care, the number of patients with pressure injuries must be accurately determined. Doing so will require careful attention to prevalence and incidence data. The data represent the percentage of patients with pressure injuries among all those surveyed in a setting (prevalence) and the percentage of patients who developed pressure injuries after admission to the setting (incidence).

In 1989, at its first consensus conference, NPIAP brought attention to the pressure injury problem in the United States by reporting prevalence and incidence data.[71] The group set a national goal to reduce the incidence of pressure injuries by 50% by the year 2000.[71] During the next decade, NPIAP engaged in an active program to improve pressure injury practice through education, research, and public policy.

At the close of the 20th century, NPIAP assessed the progress toward this goal through its Pressure Ulcers Challenge 2000 project. This 2-year project included a Medline database search for all articles on pressure ulcer incidence and prevalence published and indexed between January 1, 1990, and December 31, 2000. More than 300 studies were found. Pressure injury incidence and prevalence data were analyzed across care settings and in specific populations, such as people with spinal cord injuries, elderly patients, infants and children, patients with hip fractures, people of color, and those at the end of life receiving palliative or hospice care.[141] The Van Gilder et al. study[142] looked at “918,621 patients in the United States; data collection spanned from 2006 to 2015. The overall prevalence (OP) of PI in all facilities declined from 13.5% (2006) to 9.3% (2015). Facility-acquired prevalence (FAP) declined from 6.2% (2006) to a range of 3.1% to 3.4% (2013–2015). Acute care OP was 13.3% in 2006 and

declined to a range of 8.8% to 9.3% (2012–2015). FAP in acute care declined from 6.4% (2006) to 2.9% in 2015, with 2008–2009 showing the most aggressive decline. Long-term acute care (LTAC) had the highest OP at 32.9% in 2006; it declined to 28.8% in 2015. The LTAC-FAP was 9.0% in 2006; it declined to 5.6% in 2015. Recently, the long-term care (LTC) FAP rose from 3.8% in 2013 to 5.4% in 2015. The rehabilitation facility FAP was 2.6% to 2.8% over the last 3 years. Average patient age declined in all care settings with the exception of LTAC and LTC. Braden Scale risk scores remained constant and weight increased in all care settings with the exception of LTC."[142]

Many positive developments in the prevention and treatment of pressure injuries have occurred over the past decade, including development of evidence-based practice guidelines, standardization of risk assessment, and improved technologies for prevention and treatment.[1]

Most pressure injuries, regardless of setting, are partial thickness (stages 1 and 2) and are located on the sacrum or coccyx.[142] Heels are the second most common location. The proportion of DTPI pressure injuries has increased threefold to 9% in 2009.[143] The heels (41%), sacrum (19%), and buttocks (13%) are the locations for 73% of all DTPI. This is in a different order than the most frequent locations for all pressure injuries (sacrum, heels, etc.).[143]

Prevalence and Incidence Definitions and Formulas

Lack of clarity and consistency in definitions and calculation formulas impede our understanding of pressure ulcer prevalence and incidence. Standardization of definitions and formulas will enhance comparability of data among future studies. NPIAP recommends the adoption of consistent definitions and formulas for determining pressure ulcer prevalence and incidence.[1]

EPUAP/NPIAP/PPPIA suggests that prevalence should be defined as “the proportion/percentage of individuals in a defined population who have a medical condition at a specified point in time.[1] Suggested standard formulas for obtaining prevalence are[1]

Formulas to calculating prevalence and incidence

Formula for calculating point prevalence (%)

$$\frac{\text{Number of individuals with pressure injury at a specific point in time}}{\text{Total number of individuals in the study population at a specific point in time}} \times 100$$

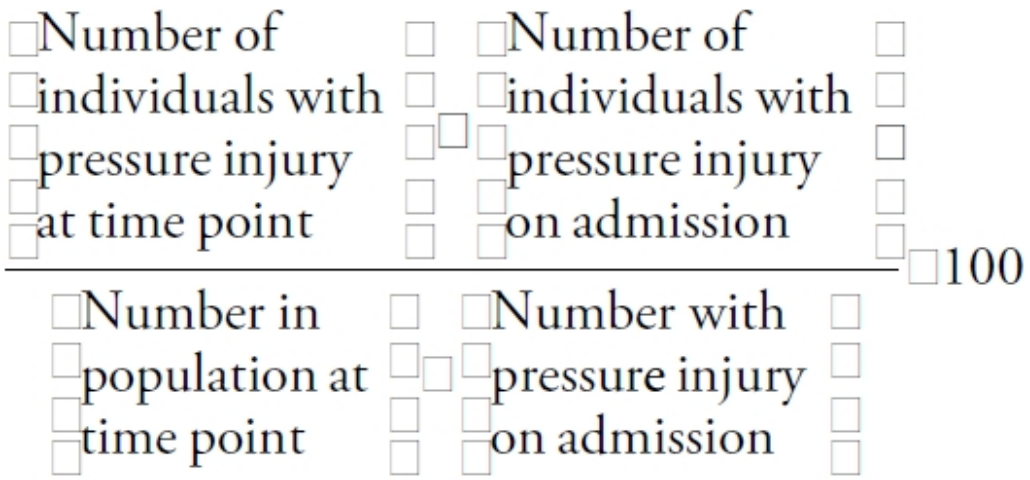
Formula for calculating cumulative incidence (%)

$$\frac{\text{Number of individuals with pressure injury during a specific time period}}{\text{Total number of individuals in the study population over a specific point in time}} \times 100$$

Formula for calculating incidence density of individuals per time (%)

$$\frac{\text{Number individuals developing a new pressure injury}}{\text{Total patient days without a pressure injury}}$$

Formula for calculating faculty-acquired pressure injury rate (%)



EPUAP/NPIAP/PPPIA recommends using the following definition of "Incidence: proportion or rate of occurrence of a given medical condition in a population within a specified period of time."[1] Several approaches to measuring incidence have been used. NPIAP defines cumulative incidence as the "proportion of a specified population that develops a new pressure injury within a specified time period (usually weeks or months)."[1]

Another way to calculate incidence is incidence density as it accounts for length of stay and therefore may be more useful in LTC facilities where residents may be there for months or even years.[1] Incidence density is calculated as the number of individuals developing a new pressure injury divided by total patient days without a pressure injury per 1,000 patient-days.[1] Calculate this by using the following suggested formula (see formula above).

Operating Room

Since CMS stopped reimbursing hospitals at a higher rate for pressure injuries that occur during hospitalization, much attention has been placed on reducing the incidence of pressure injuries during the perioperative experience. Elements of a pressure injury prevention protocol are adapted for the special needs of perioperative patients including using additional support surfaces such as facial pads to off-load pressure points on the face and other parts of the body when the patient is in the prone position. One hospital was able to reduce pressure injuries in their surgical patients from 7.3% to 1.3%.[144] This was accomplished by educating the operative staff and using a special colored risk band to identify patients with longer operating times as being at risk for pressure injuries.

Competencies and Curriculum

Accurate and current knowledge is essential for clinicians to prevent and treat pressure injuries. Nurses want more education on wounds.[93,145] Pressure injury knowledge varies among members of the wound care team. For example, physicians and residents in three studies were found to have low levels of pressure injury knowledge.[146–149] Some nurses believe that their basic education is insufficient regarding wound care.[83–93] Nurses want more education on wounds.[93,145,150] What's more, high pressure injury prevalence rates have been linked to poor knowledge.[151–154]

As knowledge about pressure injuries has increased, their occurrence has decreased across care settings.[153,154] An early initiative to decrease pressure injuries was the New Jersey Hospital Associations "No Ulcers" project[153] AHRQ pilot tested a pressure injury tool kit to assist hospitals in decreasing the incidence of pressure injury.[154] Certification has made a difference in pressure ulcer knowledge. In one study, nurses who had received any wound care certification from the WOCN Society, the American Academy of Wound Management, or the National Alliance of Wound Care had higher scores on a standardized 47-item pressure injury test.[155]

Suggestions to improve both pressure injury prevention practice and the characteristics of pressure injury incidence reduction initiatives have been reported in the literature.[156–159] For example, after introduction of the Canadian Association of Wound Care Pressure Ulcer Awareness Program, the pressure injury prevalence and incidence rates in Canada decreased by 57% and 71%, respectively.[159] In long-term care, Horn and colleagues achieved a 33% reduction in pressure ulcers in seven facilities by implementing a real-time optimal care plan for nursing home quality initiative in which the number of documentation forms was reduced from 6.2 to 2.8.[69] Citing the limited literature on pressure injury in patients undergoing procedures in diagnostic and interventional ancillary units such as radiology, renal dialysis, and cardiac and vascular procedure laboratories, Messer[160] stresses the importance of focusing on

prevention in these areas. Another PI competency checklist for direct care providers and nursing managers has also been published.[145] A recent investigation by Moore and colleagues reports findings about wound care curriculum in entry level Doctor of Physical Therapy programs in the United States.[161]

Building knowledge about pressure injury care is vital. While many experts believe that all pressure injuries cannot be prevented,[104,162] it's been shown that education can reduce pressure injury incidence and expedite treatment.[158,163] A variety of continuing education programs, symposia, national conferences, as well as company-sponsored online learning programs exist to facilitate this knowledge building. Interactive computer-based testing by the EPUAP-endorsed ePUCLAS$_4$ pressure injury staging module[94] is just one of the resources available. The John A. Hartford Institute for Geriatric Nursing also has several one-page quick references in its "Try This" series, one of which is using the Braden Scale.[164] The NDNQI offers four online modules covering several aspects of pressure injury care in which clinicians can acquire knowledge as well as measure their learning through interactive testing.[96] With all this information available, the challenge for clinicians is to put this knowledge into practice in order to prevent and/or treat pressure injuries.

Summary

Pressure injuries a common healthcare problem throughout the world. Intensity and duration of pressure and shear exposures at the surface of the body and internally in subdermal and deep tissues, as well as the individual tissue tolerance, are the etiologic factors that lead to pressure injury development. Incorporation of clinical practice guidelines provides a basis for evidence-based pressure injury prevention and treatment practice. Results of a pressure injury risk assessment using a validated tool can serve as the foundation for developing a pressure injury prevention protocol. After determining the pressure injury category/stage and other wound characteristics, a comprehensive plan to treat the pressure ulcer that uses a combination of local wound care, débridement, moist wound healing, cleaning, and pressure relief needs to be implemented. A multidisciplinary approach to patient care that includes patient and family education as well as staff education is essential. Use of the standardized formulas as proposed by NPIAP will provide a basis for universal comparison of prevalence and incidence data. Many educational resources are available to clinicians to increase their knowledge level (the EPUAP/NPIAP/PPPIA 2019 Guideline being a pivotal one) so as to decrease pressure injury incidence and enhance treatment.

PATIENT SCENARIO

Clinical Data

Mrs. GK is an 82-year-old woman who fractured her left hip. Following open reduction and internal fixation of the fracture, she was noncompliant with ambulation during her hospitalization and developed a sacral stage 2 pressure injury as well as pneumonia. She is now being cared for in her son's home with the assistance of home care services. Her prior dietary intake was inadequate as she had lost 7 pounds in 4 weeks while hospitalized. She nibbles at her food and eats less than half of her meals. Mrs. GK is able to respond to commands. She is very reluctant to get up and walk as her "bottom

is painful," so she sits in her bed, which has a standard mattress, most of the day. She wears adult incontinence briefs because it is too difficult to get her to the commode, so her skin in the pelvic area is moist. The skin on her extremities is pale, thin, and dry.

Case Discussion

Mrs. GK has several risk factors for developing pressure injuries, including her age, poor dietary intake, recent weight loss, immobility and inactivity, and moist skin from incontinence. Her plan of care included consultation with a dietician to review innovative ways of getting protein and nutrients into her diet. Protein supplements were added to the foods she likes to ingest (coffee, soups, and puddings).

She was referred to a certified wound and incontinence nurse for management of her incontinence, pressure injury, and skin care needs. Her daughter was taught that the use of incontinence pads should be discontinued and alternative strategies such as use of wearable garments that wick away moisture should be used. Because moist skin needs less pressure to break down and Mrs. GK is immobile, a pressure redistribution mattress and chair cushion were acquired for use on her bed and chair, respectively. Mrs. GK's son was educated about the importance of frequent repositioning, including the use of pillows to secure 30-degree tilts, as well as the rationale for additional support surfaces to promote healing and prevent further tissue insult. A schedule was developed to aid implementation of home care strategies for Mrs. GK. Appropriate wound dressings were placed to support healing of the stage 2 pressure injury. Physical and occupational therapists were consulted for increased ambulation and muscle strengthening exercises. Her own concerns were taken into account of which pain control was a vital element.

Show What You Know

1. **A pressure injury is a lesion caused by:**
   A. incontinence.
   B. unrelieved pressure.
   C. heat.
   D. diabetes mellitus.

2. **A patient is dragged across the bed when transferring to a stretcher. Which one of the following forces that can contribute to pressure injury development has occurred?**
   A. Electrical stimulation
   B. Shear
   C. Friction
   D. Maceration

3. **A patient has a 2- × 3-cm sacral pressure injury that has some depth and extends into the subcutaneous tissue with some undermining; no bone is palpable or visible. There's a small amount of slough seen in one corner of the wound, but most of the wound bed is visible. Using the NPIAP staging classification system, this pressure injury is:**
   A. stage 1
   B. stage 2
   C. stage 3
   D. stage 4

4. **Which of the following Braden Scale scores for an elderly man would indicate pressure injury risk?**
   A. 23
   B. 21
   C. 19
   D. 17

5. **Which one of the following should be included in a care plan to prevent pressure injuries?**

A. Turn and reposition every 5 hours.
B. Clean skin daily using hot water and soap.
C. Encourage the patient who's confined to a chair to relieve pressure every hour.
D. Limit fluids to 10 mL/kg of body weight daily.

6. **Which one of the following parameters is *not* part of the NPIAP PUSH tool?**
   A. Depth
   B. Exudate
   C. Tissue type
   D. Length × width

7. **A mucosal pressure injury from an ET tube on the lips should be staged using the NPIAP staging system.**
   A. Yes
   B. No

Answers to these questions are found on pages 684 and 685.

References

1. European Pressure Ulcer Advisory Panel, National Pressure Injury Advisory Panel and Pan Pacific Pressure Injury Alliance. In: E. Haesler, ed. *Prevention and Treatment of Pressure Injuries/Injuries: Clinical Practice Guideline. The International Guideline 2019*. NPUAP/NPIAP/PPPIA, 2019
2. Ayello, E.A., Sibbald, R.G. Pressure injuries: Nursing Sensitive indicator or team and systems sensitive indicator. Editorial. Advances in Skin & Wound Care 32(3):199-200, 2019. Available at: https://journals.lww.com/aswcjournal/Fulltext/2019/05000/Pressure_Injuries__Nursing_Sensitive_Indicator_or.1.aspx
3. Ayello, E.A. From bedsores to global healthcare- insights from Nightingale's Notes on Nursing. In: Nightingale Notes on Nursing. What it is, and What it is Not. 160th anniversary ed. With introductions by Joyce J. Fitzpatrick and Maureen Shawn Kennedy. Wolters Kluwer, 2020:57-62.
4. Abruzzesse R. Editorial. Decubitus 1:7, 1988.
5. Panel on the Prediction and Prevention of Pressure Ulcers in Adults. Pressure Ulcers in Adults: Prediction and Prevention. Clinical Practice Guideline No. 3. AHCPR Publication No. 92-0047. Rockville, MD: Agency for Health Care Policy and Research, 1992.
6. Bergstrom, N., et al. Treatment of Pressure Ulcers in Adults. Clinical Practice Guideline Number 15. Rockville, MD: Public Health Service, U.S. Department of Health and Human Services; Agency for Health Care Policy and Research publication 95-0652, December 1994.
7. Wound, Ostomy and Continence Nurses Society-Wound Guidelines Task Force. WOCN 2016 Guideline for Prevention and Management of Pressure Injuries (Ulcers): An Executive Summary. Journal of Wound, Ostomy, and Continence Nursing 44(3):241-6, May/June 2017.
8. Registered Nurses' Association of Ontario. Assessment and Management of Pressure Injuries for the Interprofessional Team, 3rd ed. Toronto, ON: Registered Nurses' Association of Ontario, 2016.
9. Gould, L., Stuntz, M., Giovannelli, M., et al. Wound Healing Society 2015 Guidelines for Pressure Ulcers. Wound Repair and Regeneration 24(1):145-62, 2016. Available at: https://doi.org/10.1111/wrr.12396
10. Norton, L., Parslow, N., Johnston, D., et al. Best Practice Recommendations for the Prevention and Management of Pressure Injuries. Foundations of Best Practice for Skin and Wound Management. Wounds Canada. 2018.
11. Padula, W., Delarmente, B.A. The National Cost of Hospital-Acquired Pressure Injuries in the United States. International Wound Journal 16(3):634-640, January 1, 2019. https://doi.org/10.1111/iwj/13071.
12. Coleman, S., Nixon, J., Keen, J. A New Pressure Ulcer Conceptual Framework. Journal of Advanced Nursing 70(10):2222-34, 2014.

13. Coleman, S., Gorecki, C., Nelson, E.A., et al. Patient Risk Factors for Pressure Ulcer Development: Systematic Review. International Journal of Nursing Studies 50:974-1003, 2013.
14. Delmore, B.A., Lebovits, S., Suggs, B., et al. Risk Factors Associated with Heel Pressure Injuries in Hospitalized Patients. Journal of Wound, Ostomy, and Continence Nursing 42(3):242-8, 2015.
15. Delmore, B., Ayello, E.A., Smith, D., et al. Refining Heel Pressure Injury Risk Factors in the Hospitalized Patient. Advances in Skin & Wound Care 32(11):512-9, 2019.
16. Berke, C.T. Pathology and Clinical Presentation of Friction Injuries: Case Series and Literature Review. Journal of Wound, Ostomy, and Continence Nursing 42(1):47-61, 2015.
17. Berke CT. Friction Injury Versus Deep Tissue Injury: Level of Tissue Involvement: A Comparison of 2 Cases. Journal of Wound, Ostomy, and Continence Nursing 46(6):539-42, 2019.
18. Kottner, J., Balzer, K., Dassen, T., et al. “Pressure Ulcers: A Critical Review of Definitions and Classifications.” Ostomy/Wound Management 55(9): 22-9, 2009.
19. Gefen, A. “Deep Tissue Injury from a Bioengineering Point of View.” Ostomy/Wound Management 55(4):26-36, 2009.
20. Van Gilder, C., Amlung, S., Harrison, P., et al. “Results of the 2008-2009 International Pressure Ulcer Prevalence Survey and a 3-year, Acute Care, Unit-specific Analysis.” Ostomy/Wound Management 55(11):39-45, 2009.
21. Cuddigan, J., Ayello, E.A., Black, J. “Saving Heels in Critically Ill Patients.” WCET Journal 28(2):16-24, 2008.
22. Kayser, S.A., VanGilder, C.A., Ayello, E.A., et al. Prevalence and Analysis of Medical Device-Related Pressure Injuries from the International Pressure Ulcer Prevalence Survey. Advances in Skin & Wound Care 31(6):276-85, 2018.
23. Apold, J., Rydrych, D. Preventing Device-related Pressure Ulcers: Using Data to Guide Statewide Changes. Journal of Nursing Care Quality 27(1):28-34, 2012.
24. Black, J.M., Cuddigan, J.E., Walko, M.A., et al. “Medical Device Related Pressure Ulcers in Hospitalized Patients.” International Wound Journal 7(5):358-65, October 2010.
25. Mucosal pressure ulcers—An NPUAP position statement. August 2008. Available at: http://www.npuap.org/wp-content/uploads/2012/01/Mucosal_Pressure_Ulcer_Position_Statement_final.pdf. Last accessed December 5, 2019.
26. Delmore, B.A., Ayello, E.A. Pressure Injuries Caused by Medical Devices and Other Objects: A Clinical Update. The American Journal of Nursing 117(12):36-45, 2017.
27. Norton, D., et al. An Investigation of Geriatric Nursing Problems in Hospital. London, UK: National Corporation for the Care of Old People, 1962.

28. Bergstrom, N., et al. “The Braden Scale for Predicting Pressure Sore Risk.” Nursing Research 36(4):205-10, July-August 1987.
29. Waterlow, J. “Pressure Sores: A Risk Assessment Card.” Nursing Times 81(48):49-55, November 27-December 3, 1985.
30. Ayello, E.A., Braden, B. “How and Why to do Pressure Ulcer Risk Assessment.” Advances in Skin & Wound Care 15(3):125-32, May-June 2002.
31. Chou, R., Dana, T., Bougatsos, C., et al. *Pressure Ulcer Risk Assessment and Prevention: Comparative Effectiveness. Comparative Effectiveness Review No. 87. Prepared by Oregon Evidence-based Practice Center under Contract No. 290-2007-10057-I. AHRQ Publication No. 12*(13)-*EHC148-EF*. Rockville, MD: Agency for Healthcare Research and Quality, May 2013. Available at: http://www.effectivehealthcare.ahrq.gov/reports/final.cfm
32. García-Fernández, F.P., Pancorbo-Hidalgo, P.L., Soldevilla Agreda, J.J. Predictive Capacity of Risk Assessment Scales and Clinical Judgment for Pressure Ulcers: A Meta-analysis. Journal of Wound, Ostomy, and Continence Nursing 41(1):24-34, 2014.
33. Ayello, E.A., Braden, B. “Why is Pressure Ulcer Risk Assessment so Important?” Nursing 31(11):75-9, November 2001.
34. Braden, B. “Translating the Braden Scale for Predicting Pressure Sore Risk: The Challenge Continues.” WCET Journal 30(2):29-33, 2010.
35. Bradenscale. Braden Scale for Predicting Pressure Sore Risk. Available at: http://www.bradenscale.com. Accessed December 5, 2014.
36. Bergstrom, N., Braden, B. “A Prospective Study of Pressure Sore Risk Among Institutionalized Elderly.” Journal of the American Geriatric Society 40(8):747-58, August 1992.
37. Bergstrom, N., et al. “Predicting Pressure Ulcer Risk: A Multisite Study of the Predictive Validity of the Braden Scale.” Nursing Research 47(5):261-9, September-October 1998.
38. Carr, D., Benoit, R. “The Role of Interventional Patient Hygiene in Improving Clinical and Economic Outcomes.” Advances in Skin & Wound Care 22(2):74-8, 2009.
39. Brindle, C.T. “Outliers to the Braden Scale: Identifying High-risk ICU Patients and the Results of Prophylactic Dressing Use.” World Council of Enterostomal Therapists Journal 30(1):11-8, 2010.
145. Black, J., Alves, P., Brindle, C.T., et al. “Use of Wound Dressings to Enhance Prevention of Pressure Ulcers Caused by Medical Devices.” International Wound Journal 12(3):322-7, 2015.
146. Philbin, S. “Pressure Ulcer Management Using Sodium Carboxymethylcellulose Hydrofiber(R) Foam Dressings.” Ostomy/Wound Management 59:10-2, 2013.
147. Bouza, C., Saz, Z., Munoz, A., et al. “Efficacy of Advanced Dressings in the Treatment of Pressure Ulcers: A Systematic Review.” Journal of Wound Care 14:193-9, 2005.

152. Santamaria, N., Gerdtz, M., Sage, S., et al. “A Randomised Controlled Trial of the Effectiveness of Soft Silicone Multi-Layered Foam Dressings in the Prevention of Sacral and Heel Pressure Ulcers in Trauma and Critically Ill Patients: The Border Trial.” International Wound Journal 12(3):302-8, 2015.
153. Santamaria, N., Liu, W., Gerdtz, M., et al. “The Cost-Benefit of Using Soft Silicone Multilayered Foam Dressings to Prevent Sacral and Heel Pressure Ulcers in Trauma and Critically Ill Patients: A Within-Trial Analysis of the Border Trial.” International Wound Journal 12(3):344-50, 2015.
148. Moore, Z.E., Webster, J. “Dressings and Topical Agents for Preventing Pressure Ulcers.” The Cochrane Database of Systematic Reviews (8):CD009362, 2013.
149. Stephen-Haynes, J. “The Role of Barrier Protection and Pressure Ulcer Prevention.” British Journal of Nursing 22:S52, S4-S8, 2013.
150. Chaiken, N. “Reduction of Sacral Pressure Ulcers in the Intensive Care Unit Using a Silicone Border Foam Dressing.” Journal of Wound, Ostomy, and Continence Nursing 39:143-5, 2012.
151. Walsh, N.S., Blanck, A.W., Smith, L., et al. “Use of a Sacral Silicone Border Foam Dressing as One Component of a Pressure Ulcer Prevention Program in an Intensive Care Unit Setting.” Journal of Wound, Ostomy, and Continence Nursing 39:146-9, 2012.
154. Torra I Bow, J.E., Rueda Lopez, J., Camanes, G., et al. “Preventing Pressure Ulcers on the Heel: A Canadian Cost Study.” Dermatology Nursing/Dermatology Nurses’ Association 21:268-72, 2009.
155. Call, E., Pedersen, J., Bill, B., et al. “Enhancing Pressure Ulcer Prevention Using Wound Dressings: What are the Modes of Action?” International Wound Journal 12(4):408-13, 2015.
156. Dealey, C., Brindle, C.T., Black, J., et al. “Challenges in Pressure Ulcer Prevention.” International Wound Journal 12(3):309-12, 2015.
157. Brindle, C.T., Wegelin, J.A. “Prophylactic Dressing Application to Reduce Pressure Ulcer Formation in Cardiac Surgery Patients.” Journal of Wound, Ostomy, and Continence Nursing 39:133-42, 2012.
158. Cubit, K., McNally, B., Lopez, V. “Taking the Pressure Off in the Emergency Department: Evaluation of the Prophylactic Application of a Low Shear, Soft Silicon Sacral Dressing on High Risk Medical Patients.” International Wound Journal 10:579-84, 2013.
159. Ohura, N., Ichioka, S., Nakatsuka, T., et al. “Evaluating Dressing Materials for the Prevention of Shear Force in the Treatment of Pressure Ulcers.” Journal of Wound Care 14:401-4, 2005.
41. Ohura, T., Takahashi, M., Ohura, N. Jr. “Influence of External Forces (Pressure and Shear Force) on Superficial Layer and Subcutis of Porcine Skin and Effects of Dressing Materials: Are Dressing Materials Beneficial for Reducing Pressure and Shear Force in Tissues?” Wound Repair and Regeneration 16:102-7, 2008.

160. Park, K.H. “The Effect of a Silicone Border Foam Dressing for Prevention of Pressure Ulcers and Incontinence-Associated Dermatitis in Intensive Care Unit Patients.” Journal of Wound, Ostomy, and Continence Nursing 41:424-9, 2014.
42. Duimel-Peeters, I.G.P., Halfens, R.J.G., Berger, M.P.F., et al. “The Effects of Massage as a Method to Prevent Pressure Ulcers: A Review of the Literature.” Ostomy/Wound Management 51(4):70-80, 2005.
43. Langemo, D., Thompson, P., Hunter, S., et al. “Heel Pressure Ulcers: Stand Guard.” Advances in Skin & Wound Care 21(6):282-92, quiz 293-95, 2008.
40. Santamaria, N., Gerdtz, M., Kapp, S., et al. A Randomised Controlled Trial of the Clinical Effectiveness of Multi-layer Silicone Foam Dressings for the Prevention of Pressure Injuries in High-risk Aged Care Residents: The Border III Trial. International Wound Journal 15(3):482-90, 2018.
161. Campbell, K.E., Woodbury, M.G., Houghton, P.E. “Implementation of Best Practice in the Prevention of Heel Pressure Ulcers in the Acute Orthopedic Population.” International Wound Journal 7(1):28-40, 2010.
44. Cereda, E., Klersy, C., Serioli, M., et al, D’Andrea, F. A Nutritional Formula Reriched with Arginine, Zinc, and Antioxidants for the Healing of Pressure Ulcers: A Randomized Trial. Annals of Internal Medicine 162(3):167-74, 2015.
45. Bergstrom, N., Horn, S.D., Rapp, M.P., et al. “A Multisite Randomized Clinical Trial in Nursing Homes.” Journal of the American Geriatrics Society 61:1705-13, 2013.
46. Rastinehad, D. “Effectiveness of a Pressure Ulcer Prevention Program in an At-risk Oncology Population.” World Council of Enterostomal Therapists Journal 28(3):12-6, 2008.
47. Yusuf, S., Okuwa, M., Shigeta, Y., et al. “Microclimate, An Development of Pressure Ulcers and Superficial Skin Changes.” International Wound Journal 12(1):40-6, 2015.
48. Coladonator, J., Smith, A., Watson, N., et al. “Prospective, Nonrandomized Controlled Trials to Compare the Effect of a Silk-like Fabric to Standard Hospital Linens on the Rate of Hospital—Acquired Pressure Ulcers.” Ostomy/Wound Management 58(10):14-31, 2012.
49. Smith, A., McNichol, L.L., Amos, M.A., et al. “A Retrospective Non-randomized Before and After Study of the Effect of Linens Constructed of Synthetic Silk-like Fabric on Pressure Ulcer Incidence.” Ostomy/Wound Management 59(4):28-34, 2013.
50. Norton, L., Coutts, P., Sibbald, R.G. “A Model for Support Surface Selection as Part of Pressure Ulcer Prevention and Management: A Preliminary Study.” World Council of Enterostomal Therapists Journal 28(3):25-9, 2008.
51. National Pressure Ulcer Advisory Panel. Support Surface Standards Initiative (S3I). Available at: http://www.npuap.org. Accessed November 18, 2019. https://npiap.com/page/S3I
52. Horn, S.D., Sharkey, S.S., Hudak, S., et al. “Pressure Ulcer Prevention in Long Term Care Facilities: A Pilot Study Implementing Standardized Nurse Aide

Documentation and Feedback Reports." Advances in Skin & Wound Care 23(3):120-31, 2010.
53. Milne, C.T., Trigilia, D., Houle, T.L., et al. "Reducing PU Prevalence Rates in the LTC Setting." Ostomy/Wound Management 55(4):50-9, 2009.
54. National Pressure Ulcer Advisory Panel. "Pressure Ulcers Prevalence, Cost, and Risk Assessment: Consensus Development Conference Statement." Decubitus 2(2):24-8, May 1989.
55. Shea, J.D. "Pressure Sores: Classification and Management." Clinical Orthopaedics and Related Research 112:89-100, October 1975.
56. International Association of Enterostomal Therapy (IAET). "Dermal Wounds: Pressure Sores. Philosophy of the IAET." Journal of Enterostomal Therapy 15(1): 9-15, January-February 1988.
57. National Pressure Ulcer Advisory Panel. "Pressure Ulcer Stages Revised by NPUAP." February 2007. Available at: http://www.npuap.org/pr2.htm. Accessed December 5, 2014.
58. National Pressure Ulcer Advisory Panel. "National Pressure Ulcer Advisory Panel Position Paper on Staging Pressure Ulcers." Available at: http://www.npuap.org/NPUAP_position_on_staging%20final%5B1%5D.pdf. Accessed December 5, 2014.
59. Lyder, C.H., Krasner, D.L., Ayello, E.A. "Clarification from the American Nurses Association on the Nurse's Role in Pressure Ulcer Staging." Advances in Skin & Wound Care 23(1):8-10, 2010.
60. Patton, R.M. "Is Diagnosis of Pressure Ulcers Within an RN's Scope of Practice?" American Nurse Today 5(1):20, 2010.
61. Ayello, E.A., Lyder, C.H. "Pressure Ulcers in Persons of Color: Race and Ethnicity." In: J. Cuddigan, et al., eds. Pressure Ulcers in America: Prevalence, Incidence, and Implications for the Future. Reston, VA: National Pressure Ulcer Advisory Panel, 2001.
62. Van Gilder, C., MacFarlane, G.D., Meyer, S. "Results of Nine International Pressure Ulcer Prevalence Surveys: 1989 to 2005." Ostomy/Wound Management 54(2):40-54, 2008.
63. Lyder, C.H., et al. Validating the Braden Scale for the Prediction of Pressure Ulcer Risk in Blacks and Latino/Hispanic Elders: A Pilot Study." Ostomy/Wound Management 44(Suppl 3A):42S-9, March 1998.
64. Lyder, C.H., et al. "The Braden Scale for Pressure Ulcer Risk: Evaluating the Predictive Validity in Black and Latino/Hispanic elders." Applied Nursing Research 12(2):60-8, May 1999.
65. Harms, S., Bliss, D.Z., Garrard, J., et al. Prevalence of Pressure Ulcers by Race and Ethnicity for Older Adults Admitted to Nursing Homes. Journal of Gerontological Nursing 40(3):20-6, 2014.
66. Bliss, D.Z., Gurvich, O., Savik, K., et al, et al. Are there Racial-ethnic Disparities in time to Pressure Ulcer Development and Pressure Ulcer Treatment in Older Adults after Nursing Homer Admission? Journal of Aging and Health 27(4):571-93, June 2015.

67. Sprigle, S., et al. “Clinical Skin Temperature Measurement to Predict Incipient Pressure Ulcers.” Advances in Skin & Wound Care 14(3):133-7, May-June 2001.
68. Rosen, J., Mittal, V., Degenholtz, H., et al. Pressure Ulcer Prevention in Black And White Nursing Home Residents: A QI Initiative of Enhanced Ability, Incentives, and Management Feedback. Advances in Skin & Wound Care 195:262-9, 2006.
69. Sieggreen, M. Pressure Ulcers in Persons of Color: Race and Ethnicity. In: B. Pieper, ed. with the National Pressure Ulcer Advisory Panel (NPUAP). Pressure Ulcers: Prevalence, Incidence, and Implications for the Future. Washington, DC: NPUAP, 2012:141-5.
71. Ayello, E.A., Baranoski, S., Salati, D.S. “Best Practices in Wound Care Prevention and Treatment.” Nursing Management 37(9):42-8, 2006.
129. Zulkowski, K., Ayello, E.A. “Urban and Rural Nurses’ Knowledge of Pressure Ulcers in the USA.” World Council of Enterostomal Therapists Journal 25(3):24-30, 2005.
130. Ayello, E.A., Baranoski, S., Salati, D.S. “A Survey of Nurses’ Wound Care Knowledge.” Advances in Skin & Wound Care 18(5):268-75, 2005.
131. Ayello, E.A., Baranoski, S., Salati, D.S. “Wound Care Survey Report.” Nursing 35(6):36-45, 2005.
84. Ayello, E.A., Baranoski, S. “2014 Survey Results: Wound Care and Prevention.” Advances in Skin & Wound Care 27(8):371-80, 2014.
80. Ayello, E.A., Baranoski, S., Jicman, W.H. Results of the 2019 Skin and Wound Survey. Nursing 2020, In press.
81. Delmore, B., Ayello, E.A. Pressure Injuries Caused by Medical Devices and Other Objects: A Clinical Update. The American Journal of Nursing 117(12): 36-45, 2017.
82. Beeckman, D.; European Pressure Ulcer Advisory Panel. PuClas4 eLearning Module. University Centre for Nursing & Midwifery and European Pressure Ulcer Advisory Panel, 2017.
83. National Database of Nursing Quality Indicators. Pressure Ulcer Training. Available at: https://www.nursingquality.org/ndnqipressureulcertraining/default.aspx. Accessed December 5, 2014.
70. Aydin, A.K., Karadağ, A. “Assessment of Nurses’ Knowledge and Practice in Prevention and Management of Deep Tissue Injury and Stage I Pressure Ulcer.” Journal of Wound, Ostomy, and Continence Nursing 37(5):487-94, 2010.
72. Preston, A., Rao, A., Strauss, R., et al. Deep Tissue Injury: A Clinical Review. The American Journal of Nursing 117(5):50-7, May 2017. doi: 10.1097/01.NAJ.0000516273.66604.c7.
73. Stewart, T.P., Magnano, S.J. “Burns or Pressure Ulcers in the Surgical Patient?” Decubitus 1(1):36-40, 1988.

74. Black, J. “Deep Tissue Injury: An Evolving Science.” Ostomy/Wound Management 55(2):4, 2009.
75. Honaker, J., Brockopp, D., Moe, K. Development and Psychometric Testing of the Honaker Suspected Deep Tissue Injury Severity Scale. Journal of Wound, Ostomy, and Continence Nursing 41(3):238-41, 2014.
76. Honaker, J., Brockopp, D., Moe, K. “Suspected Deep Tissue Injury Profile: A Pilot Study.” Advances in Skin & Wound Care 27(3):133-40, 2014.
77. Quintavalle, P.R., Lyder, C.H., Mertz, P.J., et al. “Use of High-resolution, High-frequency Diagnostic Ultrasound to Investigate the Pathogenesis of Pressure Ulcer Development.” Advances in Skin & Wound Care 19(9):498-505, 2006.
78. Koerner, S., Adams, D., Harper, S.L., et al. Use of Thermal Imaging to Identify Deep-Tissue Pressure Injury on Admission Reduces Clinical and Financial Burdens of Hospital-Acquired Pressure Injuries. Advances in Skin & Wound Care 32(7):312-20, 2019.
79. Edsberg, L., Langemo, D., Baharestani M.M., et al. “Unavoidable Pressure Injury: State of the Science and Consensus Outcomes.” Journal of Wound, Ostomy, and Continence Nursing 41(4):313-34, 2014.
85. Langemo, D.K., Brown, G. “Skin Fails Too: Acute, Chronic, and End-stage Skin Failure.” Advances in Skin & Wound Care 19(4):206-11, 2006.
86. Ayello, E.A., Levine, J.M., Langemo, D., et al. Reexamining the Literature on Terminal Ulcers, SCALE, Skin Failure, and Unavoidable Pressure Injuries. Advances in Skin & Wound Care 32(3):109-21, 2019.
87. CAWC Institute for Wound Management and Prevention. Level 1 Workbook: Putting Knowledge into Practice—Knowledge Learning. Toronto, Canada: Canadian Association of Wound Care, 2010.
88. Pieper, B., Langemo, D., Cuddigan, J. “Pressure Ulcer Pain: A Systematic Literature Review and National Pressure Ulcer Advisory Panel White Paper.” Ostomy/Wound Management 55(2):16-31, 2009.
89. Harris, C., Bates-Jensen, B., Parslow, N., et al. Wound Care: Bates-Jensen Wound Assessment Tool: Pictorial Guide Validation Project. Journal of Wound, Ostomy, and Continence Nursing 37(3):253-9, June 2010.
90. NPUAP PUSH Task Force. “Pressure Ulcer Scale for Healing: Derivation and Validation of the PUSH Tool.” Advances in Wound Care 10(5):96, September-October 1997.
91. Bates-Jensen B. Bates-Jensen Wound Assessment Tool. 2001. [cited September 2019]. Available at: http://wwwwoundcare.ca/Uploads/ContentDocuments/ BWAT.pdf
92. National Pressure Ulcer Advisory Panel. “Pressure Ulcer Scale for Healing (PUSH) Version 3.0.” Available at: http://www.npuap.org/page/PUSHTool
93. Berlowitz, D.R., Ratliff, C., Cuddigan, J., et al.; the National Pressure Ulcer Advisory Panel. “The PUSH Tool: A Survey to Determine Its Perceived Usefulness.” Advances in Skin & Wound Care 18(9):480-3, 2005.
94. Choi, E.P., Chin, W.Y., Wan, E.Y., et al. Evaluation of the Internal and External Responsiveness of the Pressure Ulcer Scale for Healing (PUSH) Tool for

Assessing Acute and Chronic Wounds. Journal of Advanced Nursing 72(5):1134-43, 2016.
95. Wallenstein, S., Brem, H. “Statistical Analysis of Wound-healing Rates for Pressure Ulcers.” The American Journal of Surgery 188(1A Suppl):73S-8, July 2004.
96. Jessup, R.L. “What is the Best Method for Assessing the Rate of Wound Healing? A Comparison of 3 Mathematical Formulas.” Advances in Skin & Wound Care 19(3):138, 140-42, 145-46, 2006.
97. Schubert, V., Zander, M. “Analysis of the Measurement of Four Wound Variables in Elderly Patients with Pressure Ulcers.” Advances in Wound Care 9(4):29-36, 1996.
98. Xakellis, G., Frantz, R.A. “Pressure Ulcer Healing: What Is It? What Influences It? How Is It Measured?” Advances in Wound Care 10(5):20-6, 1997.
99. Dyson, M., Lyder, C. “Wound Management Physical Modalities.” In: M. Morison, ed. The Prevention and Treatment of Pressure Ulcers. Edinburgh, UK: Harcourt Brace/Mosby International, 2001.
100. Sibbald, R.G., Woo, K.Y., Ayello, E.A. “Healing Chronic Wounds: DIM Before DIME Can Help.” Ostomy/Wound Management 12(Suppl), April 2009.
101. Robson, M.C., Heggers, J.P. “Bacterial Quantification of Open Wounds.” Military Medicine 134(1):19-24, February 1969.
102. Barr, J.E. “Principles of Wound Cleansing.” Ostomy/Wound Management 41(Suppl 7A):15S-22, August 1995.
103. Moore, Z.E., Cowman, S. “Wound Cleansing for Pressure Ulcers.” The Cochrane Database of Systematic Reviews (3):CD004983, 2013.
104. Hellewell, T.B., et al. “A Cytotoxicity Evaluation of Antimicrobial Wound Cleansers.” Wounds 9(1): 15-20, 1997.
105. Wilson, J.R., Mills, J.G., Prather, I.D., et al. “A Toxicity Index of Skin and Wound Cleansers Used on in Vitro Fibroblasts and Keratinocytes.” Advances in Skin & Wound Care 18(7):373-8, 2005.
106. Rodeheaver, G.T., et al. “Wound Cleansing by High Pressure Irrigation.” Surgery, Gynecology & Obstetrics 141(3):357-62, September 1975.
107. Stevenson, T.R., et al. “Cleansing the Traumatic Wound by High Pressure Syringe Irrigation.” JACEP 5(1):17-21, January 1976.
108. Bhaskar, S.N., et al. “Effect of Water Lavage on Infected Wounds in the Rat.” Journal of Periodontology 40(11):671-2, November 1969.
109. Yarkony, G.M., ed. Pressure Ulcers: Medical Management. Spinal Cord Injury Medical Management and Rehabilitation. Gaithersburg, MD: Aspen, 1994.
110. Dolychuck, K.N. “Debridement.” In: D. Krasner, et al., eds. Chronic Wound Care: A Clinical Source Book for Health Care Professionals, 3rd ed. Wayne, PA: HMP Communications, 2001.
111. Galpin, J.E., et al. “Sepsis Associated with Decubitus Ulcers.” American Journal of Medicine 61(3):346-50, September 1976.
162. Kim, Y.C., et al. “Efficacy of Hydrocolloid Occlusive Dressing Technique in Decubitus Ulcer Treatment: A Comparative Study.” Yonsei Medical Journal

37(3):181-5, June 1996.
112. Bolton, L.L., et al. “Quality Wound Care Equals Cost-effective Wound Care: A Clinical Model.” Advances in Wound Care 10(4):33-8, July-August 1997.
113. Saydak, S. “A Pilot of Two Methods for the Treatment of Pressure Ulcers.” Journal of Enterostomal Therapy 7(3):139-42, May-June 1990.
114. Lyder, C.H. “Examining the Cost-effectiveness of Two Methods for Healing Stage II Pressure Ulcers in Long-term Care.” Unpublished data, 2010.
115. Kloth, L.C., McCulloch, J. “Promotion of Wound Healing with Electrical Stimulation.” Advances in Wound Care 9(5):42-5, September-October 1996.
116. Polak, A., Kloth, L.C., Blaszczal, E., et al. The Efficacy of Pressure Ulcer Treatment with Cathodal and Cathodal-anodal High Voltage Monophasic Pulsed Current: A Prospective, Randomized, Controlled Trial. Physical Therapy 97 (8):777-89, 2017.
117. Polak, A., Kloth, L.C., Blaszczal, E., et al. Evaluation of the Healing Progress of Pressure Ulcers Treated with Cathodal High-voltage Monophasic Pulsed Current: Results of a Prospective, Double-blind, Randomized Clinical Trial. Advances in Skin & Wound Care 29(10):447-59, 2016.
118. Gardner, S.E., et al. “Effect of Electrical Stimulation on Chronic Wound Healing: A Meta-analysis.” Wound Repair and Regeneration 7(6):495-503, November-December 1999.
119. Mustoe, T.A., et al. “A Phase II Study to Evaluate Recombinant Platelet-derived Growth Factor-BB in the Treatment of Stage 3 and 4 Pressure Ulcers.” Archives of Surgery 129(2):213-9, February 1994.
120. Berlowitz, D. Prevalence, Incidence, and Facility-acquired Rates. In: B. Pieper, National Pressure Ulcer Advisory Panel, eds. Pressure Ulcers: Prevalence, Incidence, and Implications for the Future. Washington, DC: NPUAP, 2012.
121. Van Gilder, C., Lachenbruch, C., Algrim-Boyle, C., et al. The International Pressure Ulcer Prevalence TM Survey: 2006-2015 A 10-Year Pressure Injury Prevalence and Demographic Trend Analysis by Care Setting. Journal of Wound, Ostomy, and Continence Nursing 44(1):1-9, 2017.
122. Van Gilder, C., MacFarlane, G.D., Harrison, P., et al. “The Demographics of Suspected Deep Tissue Injury in the United States: An Analysis of the International Pressure Ulcer Prevalence Survey 2006-2009.” Advances in Skin & Wound Care 23(6):254-61, 2010.
123. Delmore, B., Lebovits, S., Baldock, P., et al. “Pressure Ulcer Prevention Program: A Journey.” Journal of Wound, Ostomy, and Continence Nursing 38(5):505-13, 2011.
124. Ayello, E.A., Zulkowski, K., Capezuti, E., et al. Educating Nurses in the United States About Pressure Injuries. Advances in Skin & Wound Care 30(2):83-94, 2017.
125. Garcia, A.D., Perkins, C., Click, C., et al. “Pressure Ulcer Education in Primary Care Residencies.” In: Ayello, E.A., Baranoski, S. eds., Research Forum: Examining the problem of pressure ulcers. Advances in Skin & Wound Care 18(4 Suppl):193-4, 2005.

126. Levine, J., Ayello, E.A., Zulkowski, K., et al. Pressure Ulcer Knowledge in Medical Residents: An Opportunity for Improvement. Advances in Skin & Wound Care 25(3):115-7, 2012.
127. Springett, J., Cowell, J., Heanet, M. “Using Care Pathways in Pressure Area Management. A pilot study.” Journal of Wound Care 8(5):227-30, 1999.
128. Khan, T., Lokhandwala, A., Talatini, H. Pressure Injury Prevention: A Shared Inter-professional Responsibility by Enhancing Pressure Injury Knowledge among Resident Physicians. WCET Journal 38(4):18-27, 2018.
132. Boxer, E., Maynard, C. “The Management of Chronic Wounds: Factors That Affect Nurses’ Decision Making.” Journal of Wound Care 8(8):409-12, 1999.
133. Lamond, D., Farnell, S. “The Treatment of Pressure Sores: A Comparison of Novice and Expert Nurses’ Knowledge, Information Use and Decision Accuracy.” Journal of Advanced Nursing 27:280-6, 1998.
134. Maylor, M., Torrance, C. “Pressure Sore Survey Part 2: Nurses Knowledge.” Journal of Wound Care 8(2):49-52, 1999.
135. Ayello, E.A., Zulkowski, K.M., Holmes, A.M., et al. New Jersey Pressure Ulcer Collaborative = Results. Nursing Outlook 58(2):e4, 2010.
136. Agency for Healthcare Research and Quality. “Preventing Pressure Ulcers in Hospitals: A Toolkit for Improving of Quality Care.” Developed by the Boston University Research Team. Available at: http://www.ahrg.gov/research/etc/pressureulcertoolkit. Accessed April 22, 2011.
137. Zulkowski, K., Ayello, E.A., Wexler, S. “Certification and Education: Do They Affect Pressure Ulcer Knowledge in Nursing?” Advances in Skin & Wound Care 20(1):34-8, 2007.
138. Lyder, C.H., Ayello, E.A. “An Annual Checkup—One Year after the Implementation of the CMS Present on Admission and Pressure Ulcers.” Advances in Skin & Wound Care 22(10):476-84, quiz 485-86, 2009.
139. Armstrong, D.G., Ayello, E.A., Capitulo, K.L., et al. “New Opportunities to Improve Pressure Ulcer Prevention and Treatment: Implications of the CMS Inpatient Hospital Care Present on Admission Indicators/Hospital-Acquired Conditions Policy—A Consensus Paper from the International Expert Wound Care Advisory Panel.” Advances in Skin & Wound Care 21(10):469-77, 2008.
140. Neiderhauser, A., VanDeusen Lukas, C., Parker, V., et al. “Comprehensive Programs for Preventing Pressure Ulcers; A Review of the Literature.” Advances in Skin & Wound Care 25(4):167-88, 2012.
141. Orsted, H.L., Rosenthal, S., Woodbury, M.G. “Pressure Ulcer Awareness and Prevention Program. A Quality Improvement Program Through the Canadian Association of Wound Care.” Journal of Wound, Ostomy, and Continence Nursing 36(2):178-83, 2009.
163. Messer, M.S. “Pressure Ulcer Risk in Ancillary Services Patients.” Journal of Wound, Ostomy, and Continence Nursing 37(2):153-8, 2010.
142. Moore, K.D., Hardin, A., VanHoose, L., et al. Current Wound Care Education in Entry-level Doctor of Physical Therapy Curricula. Advances in Skin &

Wound Care 33(1):47-52, 2020.
143. Brandeis, G.H., Berlowitz, D.R., Katz, P. “Are Pressure Ulcers Preventable? A Survey of Experts.” Advances in Skin & Wound Care 14:244, 2001.
164. Carasa, N., Polycarpe, M. Caring for the Chronically Ill Patient: Establishing a Wound Healing Program in a Respiratory Care Unit. The American Journal of Surgery 188(1A Suppl):18S-21, July 2004.
144. Ayello, E.A., for the Hartford Institute for Geriatric Nursing. “Try This Series: Predicting Pressure Ulcer Risk.” Available at: http://consultgerirn.org/uploads/File/trythis/try_this_5.pdf. Accessed December 5, 2014.

15

Venous Disease and Ulcers

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Objectives

After completing this chapter, you will be able to:

- describe the anatomy and physiology of the venous system
- describe the pathophysiology of lower extremity venous ulcers
- explain the physiology of edema formation
- state the signs and symptoms that constitute a venous assessment
- discuss vascular laboratory tests performed for patients with venous disease describe systems for classifying venous disease
- describe the components of local wound care for a patient with a venous ulcer
- describe surgical treatment for patients with venous ulcers
- identify education needs for patients with venous disease.

We gratefully acknowledge the contributions of Ronald A. Kline, MD, for his work on previous editions of this chapter.

Introduction

“Peripheral vascular disease” is a term commonly used in reference to an arterial problem, even though it includes diseases and conditions of the venous and lymphatic systems as well as the arterial system. Patients with leg ulcers may present with a combination of arterial, venous, and lymphatic disease. This chapter presents information regarding the pathogenesis and management of wounds resulting from venous insufficiency.

Scope of the Problem

Up to 50% of individuals may have some sort of vein abnormality.[1] It is estimated that between 1% and 22% of the population over age 60 suffers from lower extremity skin ulcers.[2–5] One study found the problem to be underestimated when a self-report survey indicated that high numbers of patients cared for their own ulcers without consulting a healthcare provider.[6] The principal etiology for leg ulcers in most patients is some type of peripheral vascular disease. Chronic venous disease is the seventh most common chronic disease and is the underlying cause in 95% of leg ulcers.[1,7–9] In a U.S. community health survey, 5% of adults had skin changes in the leg and more than 500,000 suffered from venous ulcers. Over 2,000,000 workdays are lost in the United States per year due to the associated morbidity of postphlebitic syndrome.[9] Although it's understood that chronic wounds have physical, financial, and psychological effects, it's difficult to measure these effects on a patient's quality of life.[10] It is also difficult to obtain accurate etiological information about leg ulcers, and in about one-third of medical records, no ulcer etiology might be documented.

Venous Anatomy and Physiology

Venous System

The venous system begins at the postcapillary level. Venules begin to coalesce, forming small veins, which again coalesce into larger veins from the periphery to a more central location. The venous system mimics the arterial system in many respects but has greater anatomic variability than the arterial tree. In the leg, the veins that course with the tibial and peroneal arteries are usually paired with numerous cross-linking branches, resulting in a retia appearance in some patients. These branches ascend along the respective arteries to form the popliteal vein, which is the first vein of significant size in the lower leg. The popliteal vein proceeds toward the head and becomes the femoral vein, commonly called the superficial femoral vein—a name that causes confusion because the vein in question is actually a deep vein. The superficial femoral vein joins the deep femoral vein to form the common femoral vein. The deep femoral vein is the deep drainage system of the thigh (Fig. 15-1).

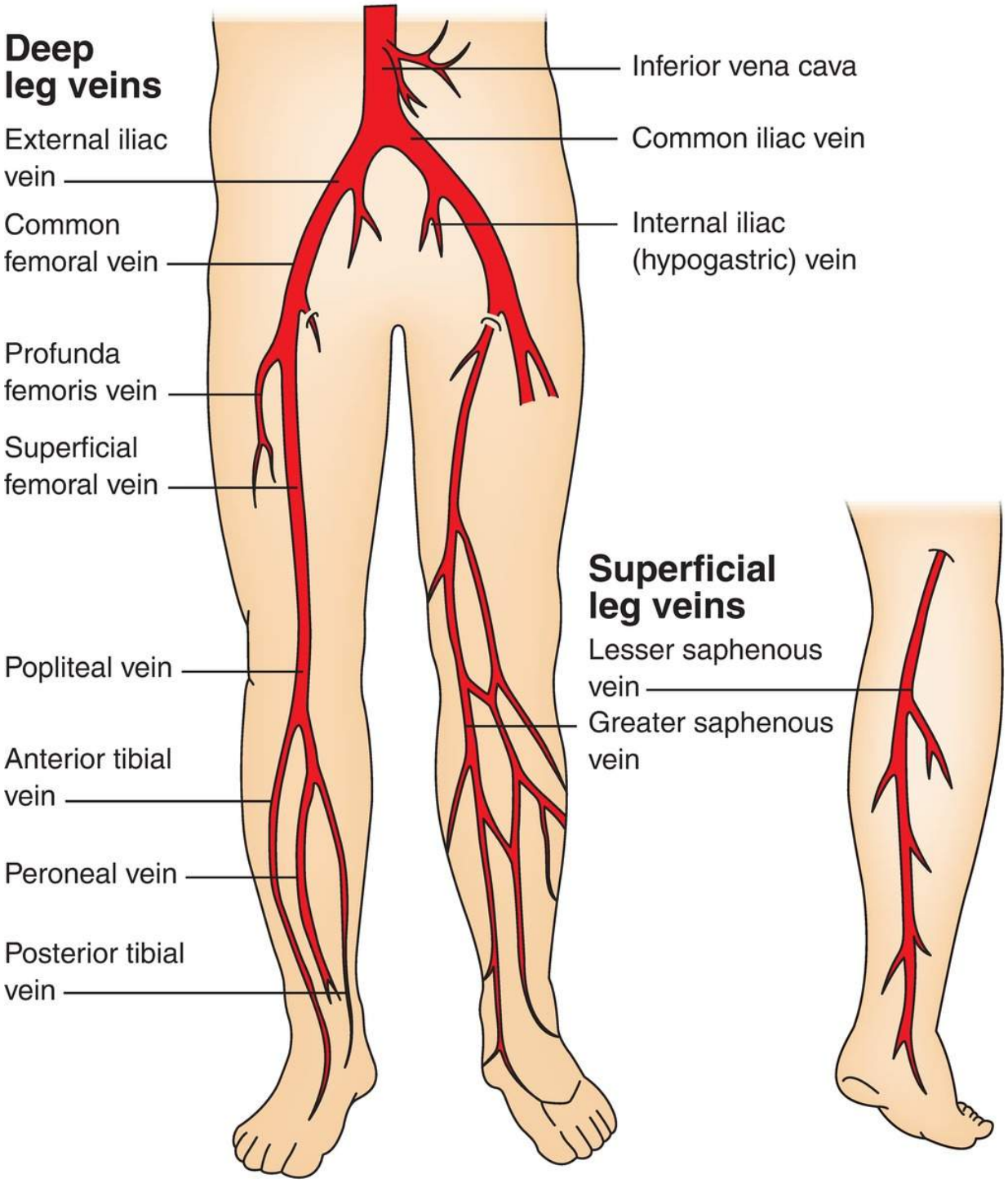

Figure 15-1. Deep and superficial venous systems. This illustration shows deep and superficial veins of the lower extremities.

Dual Venous System

The leg has a dual venous system—the deep system just described and the superficial system represented by the saphenous veins. The greater saphenous vein (GSV) courses along the medial aspect of the leg. The dorsal digital veins in the foot coalesce to form the GSV, which is found medial and anterior to the medial malleolus. It ascends in the leg through a variable course and may be bifurcated or even trifurcated. At knee level, its course becomes deeper into the saphenous compartment in relation to the skin. As it ascends the leg, it joins the common femoral vein at the fossa ovale. The lesser saphenous vein drains the posterior aspect of the calf. It perforates into the deep compartment of the calf at the level of the popliteal fossa to join the popliteal vein. As the common femoral vein ascends behind the inguinal ligament, it becomes the external iliac vein and joins the internal iliac vein to become the common iliac vein. The common iliac veins join at the level of the umbilicus and to the right of the aorta to become the inferior vena cava. The renal veins drain into the inferior vena cava. More cephalad, the hepatic veins join the inferior vena cava, which then empties into the right heart chamber.

The saphenous system is connected to the deep venous system through numerous perforator veins. Perforator veins shunt blood from the subcutaneous tissue and the greater saphenous system into the deep veins of the leg. They cross through the superficial fascia of the leg, hence their name. The location of perforator veins is somewhat variable, and some are ascribed proper names. The lowest perforator connecting the saphenous system with the deep venous system is just above the medial malleolus.

Valve Anatomy

Unidirectional valves are present in the deep and superficial venous systems and in the perforator veins. These valves are located just before bifurcation points. The GSV contains approximately six to eight valves. With rare exception, a valve is always present just below the insertion of the GSV into the common femoral vein at the fossa ovale. The orientation of the valves allows venous blood to flow from distal to proximal. Perforator veins valves are oriented to shunt blood from the lesser saphenous vein and the greater saphenous system into the deep veins of the leg.

Valve anatomy is that of a bileaflet with valve sinuses present on the lateral bases of each valve leaflet. These sinuses represent a dilation in the normal contour of the vein wall. Their function is to assist in valve closure, a passive act caused by the retrograde flow of venous blood into the sinus, thereby coapting (fitting together) the two valve leaflets. The valve leaflets are oriented parallel to the surface of the skin. This system is dependent on calf muscle contraction that forms the base of the calf muscle pump squeezing blood in a retrograde direction by creating a pressure gradient between the calf muscle and the upper leg.[11] It is the loss of valve function at various levels that results in varying degrees of venous insufficiency (Fig. 15-2). Valve function is lost under a number of disease states. Inability of the valve to coapt can also occur with over distention of the venous segment. This effectively stretches the valves apart so that they no longer come in direct contact, thereby allowing blood to reflux into the more dependent portion of the vein. Disease states that cause loss of valve function include:

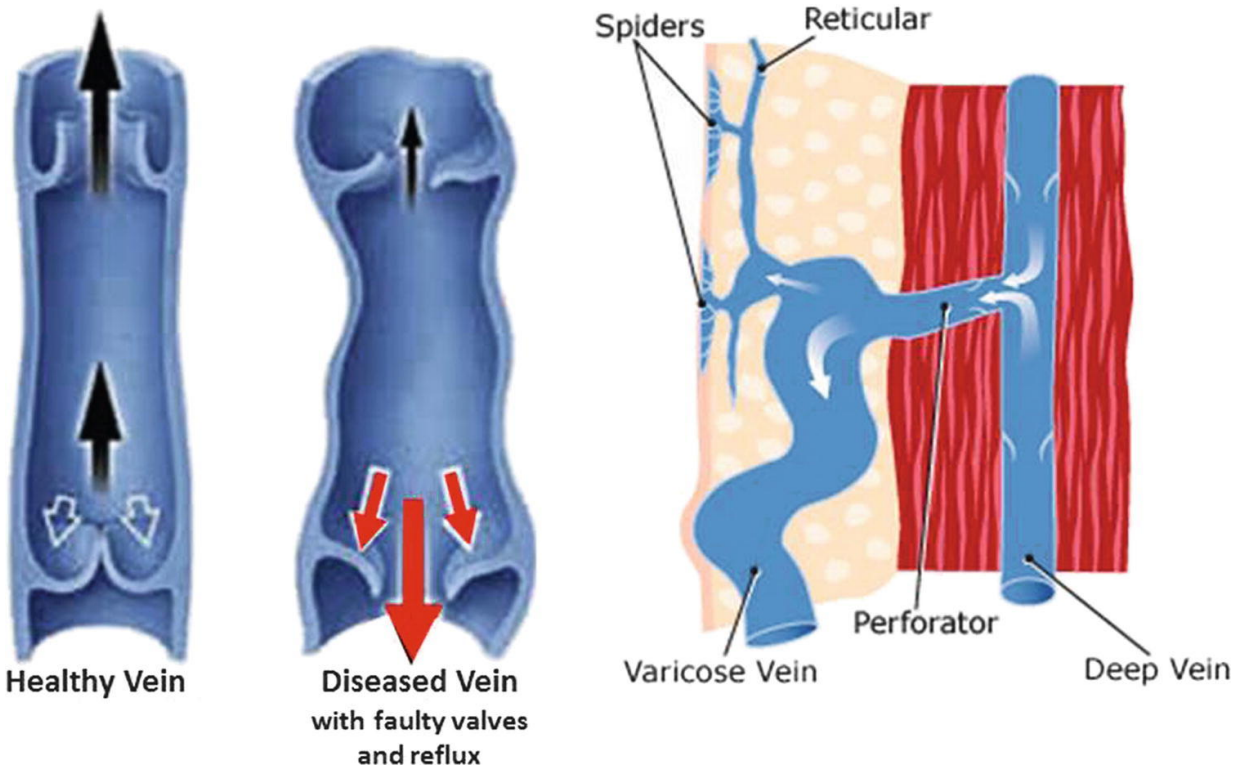

Figure 15-2. Pathophysiology of venous insufficiency. Faulty valves resulting in reflux and vein dilation and varicosity. (Courtesy J.A. Niezgoda.)

- congenital valve absence
- deep vein thrombosis
- ectasia
- phlebitis
- valve atresia
- venous engorgement
- venous hypertension
- ankle joint disease leading to stiffness.

Venous Wall Architecture

Venous wall anatomy is similar to arterial wall anatomy except that the respective laminae are thinner. The outermost layer of a vein is the adventitia. The media varies most from the arterial media. The media within a vein contains both elastic and muscular fibers, but to a much lesser degree than the arterial media. Nonetheless, a vein can contract and adjust its size to correspond to the degree of venous blood flow. The intima layer is a delicate single layer of endothelial cells.

The relatively thin media accounts for the lack of venous compliance at increased pressures. At low pressures, the venous system is fairly compliant, but when arterial pressure is reached, the venous wall becomes distended and rigid. Venoconstriction occurs in both the superficial and deep veins; the more peripheral the vein, the more readily it contracts. This reactivity is under sympathetic adrenergic control. Peripheral veins are more sensitive than central veins to this sympathetic drive. The ability of veins to relax and dilate enables the venous system to hold 75% of the total blood volume.

Loss of consciousness can occur from venous pooling in motionless lower extremities—a fate not unknown to young military recruits who must stand at attention for prolonged periods of time.

The upward flow of lower extremity venous blood, although aided by unidirectional valves and arterial pressure, is mostly dependent upon the “muscular pump.” Pedal dorsal vein pressure in the supine position should approximate that of central venous pressure. Upon assuming an erect posture, this pressure can approach 100 mm Hg. With active muscle contraction, intracompartmental pressure markedly increases, thereby causing deep veins of the leg to compress and push venous blood upward. This pressure then approaches 200 mm Hg. This is possible because the muscular compartments of the leg are enclosed by relatively rigid fascial encasement. Backflush of blood is reduced when valves are competent and reflux into the saphenous system is prevented by the unidirectional perforator valves.

Ulcer Pathophysiology

Venous Ulcers

Venous ulcers are chronic skin and subcutaneous lesions usually found on the lower extremity at the pretibial and the medial supramalleolar areas of the ankle, where the perforator veins are located. Venous ulcers were formerly known as “venous stasis” ulcers because their development was thought to be caused by blood pooled in the veins. The literature indicates that venous hypertension rather than venous stasis is both the cause of these ulcers and the reason they don’t heal.[12,13] It is difficult to restore skin integrity in the presence of chronic venous hypertension because the underlying edema must be controlled in addition to healing the ulcer.

Venous ulceration may be precipitated by deep vein thrombosis (DVT), which can remain undiagnosed for years prior to the onset of the ulcer (Fig. 15-3). It has long been thought that the natural course of lower extremity DVT is the eventual development of leg ulcers.[14]

A
B

Figure 15-3. Venogram. In this venogram, the patient's left venous valve (*B*) is intact. On the patient's right (*A*), collateral veins are present due to venous occlusion, possibly from an undiagnosed deep vein thrombosis.

Symptomatic and asymptomatic thrombi may cause long-term complications by scarring the intima and creating valvular incompetence. When the valves are rendered incompetent, blood backs into the distal veins during diastole. With loss of perforator valve function, the high intracompartmental venous pressure, which can approach 200 mm Hg during active muscle contraction, results in distention of the saphenous system. This in turn causes a cascading effect with dilation of the GSV and worsening of already compromised valvular function. The weight of the column of blood increases the pressure inside the capillaries.

Characteristics of Venous Ulcers

Venous hypertension distends the superficial veins, resulting in vein wall damage and exudation of fluid into the interstitial space, thereby causing edema of venous insufficiency. Over time, an actual leakage of red blood cells occurs through these compromised veins. As they break down, the red blood cells deposit hemosiderin into the tissues, causing a form of "internal tattooing" of the skin; the coloration is that of a brownish hue noticeable even in darkly pigmented skin (Fig. 15-4). The skin loses its normal texture and becomes somewhat shiny and subsequently sclerotic, giving a taut skin appearance in these areas with loss of skin elasticity leading to skin breakdown with minimal trauma. This is due to the resulting loss of collagen in the upper dermal layers.[14]

Figure 15-4. Hemosiderin staining. Hemosiderin staining caused the discoloration seen here in the patient's right leg from chronic venous hypertension. (Courtesy J.A. Niezgoda.)

Edema and loss of red cells into the subcutaneous tissue occur at the point of greatest gravitational pressure, the ankle. This gives rise to the pathognomonic features of chronic venous stasis, hyperpigmentation, and stocking distribution induration of the subcutaneous tissues,[15,16] the characteristics of long-standing venous insufficiency called lipodermatosclerosis. These areas are prone to subsequent ulceration or infection; extreme pruritus and excoriation are usually present, potentially aggravating the injured skin. Dermatitis due to endogenous or exogenous sources and severe allergic reactions may complicate the situation. The skin may present as itchy, erythematous, and weeping or dry and scaly (Fig. 15-5). Chemical or mechanical factors may be responsible for contact dermatitis surrounding a leg ulcer.[17]

A

Figure 15-5. (A) Venous ulcer with granulation tissue and epithelial islands and epithelial growth from the borders. **(B)** Venous ulcer with granulating base.

Another sequela of venous hypertension is irritability of the musculature. Many patients with venous insufficiency—even those in whom the condition is mild—report nocturnal leg cramps. Depolarization may occur due to fluid distention of the muscular cells, causing tetanic-like contractions of various muscle groups. Distention of veins in the subdermal plexus results in the varicosities typically seen with venous insufficiency (Fig. 15-6). The appearance of telangiectasias, commonly called “spider veins,” is the result of distention of the smaller subdermal capillary network (Fig. 15-7).

Figure 15-6. Note the presence of varicose veins in the patient's right leg.

Figure 15-7. Telangiectasias, also known as “spider veins,” are shown. (Courtesy J.A. Niezgoda.)

In some circumstances, venous aneurysms can occur due to massive dilation of the GSV and its tributaries. Further stagnation of flow in these areas in the presence of an abnormal vessel wall can result in thrombophlebitis, which worsens the venous outflow of the leg and aggravates an already deleterious condition. Thrombosis adheres to the wall of the vein, and although recanalization occurs eventually, the valves remain incompetent. In an attempt to compensate for the reduced venous return, the surrounding collateral veins dilate. Chronic edema occurs in the ankle. Increased venous pressure impedes capillary flow, decreasing oxygen available for transport from the capillaries to the tissues, and protein and red blood cells leak into the interstitial tissues. The effect is cumulative, eventually leading to tissue damage, scar formation, and, ultimately, ulceration.

Endothelium in the normal saphenous vein facilitates contraction in response to noradrenaline. In varicose veins, the endothelial-enhanced noradrenaline vasoconstriction is decreased. Endothelial damage is thought to be a possible cause of venous dilatation and subsequent varicose veins.[18]

Venous leg ulcers are also correlated with increased ambulatory venous pressures (AVP). Nicolaides[19] obtained AVP from 220 patients admitted with venous problems. He found that no patients with an AVP less than 30 mm Hg had leg ulcers, while 100% of those with AVP greater than 90 mm Hg had ulcers. The incidence of ulceration wasn't preferentially associated with either superficial or deep venous disease.

Nicolaides' study[20] suggests that AVP should be measured in patients with nonhealing venous ulcers to determine whether they may benefit from a procedure such as a venous valve transplant, which reduces the AVP to less than 30 mm Hg.

Pathogenesis of Venous Ulcers

Several theories have been proposed to explain the mechanism of venous hypertension leading to ulceration. In 1917, Homans suggested that stasis of blood in dilated veins in the skin may cause anoxic cell death, leading to ulcers. In 1929, Blalock[21] found blood oxygen content to be higher than normal in varicose veins, suggesting

that arteriovenous communications may be responsible for venous hypertension. In 1972, however, a study using radioactive macroaggregates refuted the arteriovenous shunting hypothesis.[22]

Two hypotheses—the “fibrin cuff” and “white cell trapping” theories—are relatively attempts to explain venous ulcer formation. The fibrin cuff theory states that sustained venous hypertension causes distention of dermal capillary beds, which allows plasma exudate to leak into the surrounding tissue. Fibrin precipitation in the peripapillary space forms fibrin cuffs, which impair oxygen, nutrient, and growth factor transport. The tissues undergo inflammation and fibrosis.[23] A subsequent study suggests that peripapillary fibrin is present but doesn’t influence healing of lower extremity ulcers.[24]

The white cell trapping theory states that the neutrophil aggregation in the capillaries causes lipodermatosclerosis. Increasing venous pressure is thought to reduce capillary perfusion pressure and flow rate. Low capillary flow rate initiates white blood cell adherence to the cell wall. Endothelial cells and leukocytes interact and release proteolytic enzymes, oxygen-free radicals, and lipid products. The white cells are then activated, damaging the vessel walls, increasing capillary permeability, and allowing larger molecules such as fibrinogen to exit the capillaries.[25,26]

The trap hypothesis of venous ulceration was proposed by Falanga and Eaglstein.[27] This hypothesis proposes that fibrin and other macromolecules that leak out bind or trap growth factors and other substances necessary for maintaining normal tissues and healing. There has not been any new research to support either of these theories. The resulting lower leg stain associated with lipodermatosclerosis is often misdiagnosed as bilateral cellulitis in acute flare-up situations[28] (Fig. 15-8).

Figure 15-8. Lipodermatosclerosis and hemosiderin staining.

Classifying Venous Disease

Chronic venous insufficiency (CVI) has been defined as an abnormally functioning venous system caused by venous valvular incompetence with or without associated venous outflow obstruction, which may affect the superficial venous system, the deep venous system, or both.[29] CVI can result in postphlebitic syndrome, which manifests as varicose veins and venous ulcers.

The American Venous Forum developed a descriptive system based on clinical, etiologic, anatomic, and pathophysiologic (CEAP) data to categorize the key elements in chronic venous disease.[30] The CEAP system provides an objective classification method that clarifies relationships among contributing factors and improves communication

regarding venous disease (Table 15-1). The system is subdivided into seven categories based on objective signs of chronic venous disease.[31] In 2004,[32] this system was refined to add C_0 to the clinical component to represent no visible or palpable signs of venous disease, and subclasses were also added.

Table 15-1 CEAP Classification System

The CEAP classification system for chronic venous disease consists of four elements:

- **C**linical classification
- **E**tiological classification
- **A**natomical classification
- **P**athophysiological classification

Clinical Classification (C_0–C_6)

Class	Description
0	No signs of venous disease
1	Telangiectasias or reticular veins
2	Varicose veins
3	Edema
4a	Pigmentation and eczema
4b	Lipodermatosclerosis and atrophie blanche
5	Healed venous ulcer
6	Active venous ulcer

Etiological Classification (E_C, E_P, E_S)

- Congenital (E_C)
- Primary (E_P)—with undetermined cause
- Secondary (E_S)—with known cause

Anatomical Distribution Classification (A_S, A_D, and A_P)

This element consists of classifications A_S, A_D, and A_P, and segments 1 through 18. See table below for a breakdown.

- Superficial veins (A_S)
- Deep veins (A_D)
- Perforating veins (A_P)

Segment	Classification
As	Superficial veins
1	Superficial veins Telangiectasias/reticular veins Greater (long) saphenous vein
2	• Above knee
3	• Below knee
4	• Lesser (short) saphenous
5	• Nonsaphenous
Ad	Deep veins
6	Inferior vena cava Iliac
7	• Common
8	• Internal
9	• External
10	Pelvic–gonadal, broad ligament, other femoral

11	• Common
12	• Deep
13	• Superficial
14	• Popliteal
15	Crural: anterior tibial, posterior tibial, peroneal (all paired)
Ap	Perforator veins
16	Muscular: gastrocnemial, soleal, other perforating veins
17	Thigh
18	Calf
Pathophysiological Classification (P_R, P_O) • P_R: reflux • P_O: obstruction • P_R, P_O: reflux and obstruction	

Adapted from Porter, J.M., Moneta, G.L. "Reporting Standards in Venous Disease: An Update. International Consensus Committee on Chronic Venous Disease". *Journal of Vascular Surgery* 21:635-45, 1995. Eklof, B., Rutherford, R.B., Bergan, J.J., et al. "Revision of the CEAP Classification for Chronic Venous Disorders: Consensus Statement." *Journal of Vascular Surgery* 40:1248-52, 2004. Meissner M.H., et al. "Primary Chronic Venous Disorders." *Journal of Vascular Surgery* 46:54S-67S, 2007.

C_4 was added to better define the differing severity of venous disease:

C_{4a}: Pigmentation or eczema

C_{4b}: Lipodermatosclerosis or atrophie blanche

In 2000, The American Venous Forum developed an assessment tool called the Venous Clinical Severity Score (VCSS) to supplement the CEAP tool.[33] This assessment tool was revised and updated in 2010.[34] Designed to provide more information about severe chronic venous disease, the VCSS tool consists of six descriptors—pain, varicose veins, venous edema, skin pigmentation, inflammation, and induration—that are rated 0 (absent), 1 (mild), 2 (moderate), or 3 (severe). In addition, there are four descriptors for ulcers: number of

active ulcers (0, 1, 2, or 3 or more), active ulcer duration (N/A, <3 months, >3 months but <1 year, not healed for 1 year), active ulcer size (N/A, <2 cm diameter, 2 to 6 cm diameter, >6 cm diameter), and compressive therapy (not used, intermittent use, wears most days, full compliance). This scoring system is an elaboration of the C or clinical component of the CEAP classification system. It is dynamic in that the numbers can change as the ulcers heal or the venous disease process is treated. Combining the two tools provides a method to communicate information about the ulcer in both objective and subjective terms. The VCSS and CEAP tools are expected to improve clinician's ability to follow venous treatment outcomes.

Diagnosing Venous Ulcers

Venous disease and ulcer etiology can be determined by obtaining a thorough patient history and performing a physical examination. A focused vascular history includes a clear description of the presenting complaint, past medical history for vascular and related conditions, current and previously taken medications, and risk factors. Signs and symptoms of lower extremity venous disease may include pain, tissue loss, or change in appearance or sensation. It is important to include assessment of the arterial system when evaluating venous and lymphatic disease. Adequate arterial perfusion is essential when using compression therapy for venous and lymphatic ulcers. An ankle–brachial index (ABI) is a reliable test that can be performed in any office (Fig. 15-9). An ABI should be conducted before applying any compression therapy. It may be necessary to correct the arterial problem before the ulcer can be treated. Figure 15-10 is a sample documentation tool available to be used by clinicians. Noninvasive vascular laboratory testing is used to identify the location of vascular pathology.

Figure 15-9. Ankle–brachial index (ABI) evaluation.

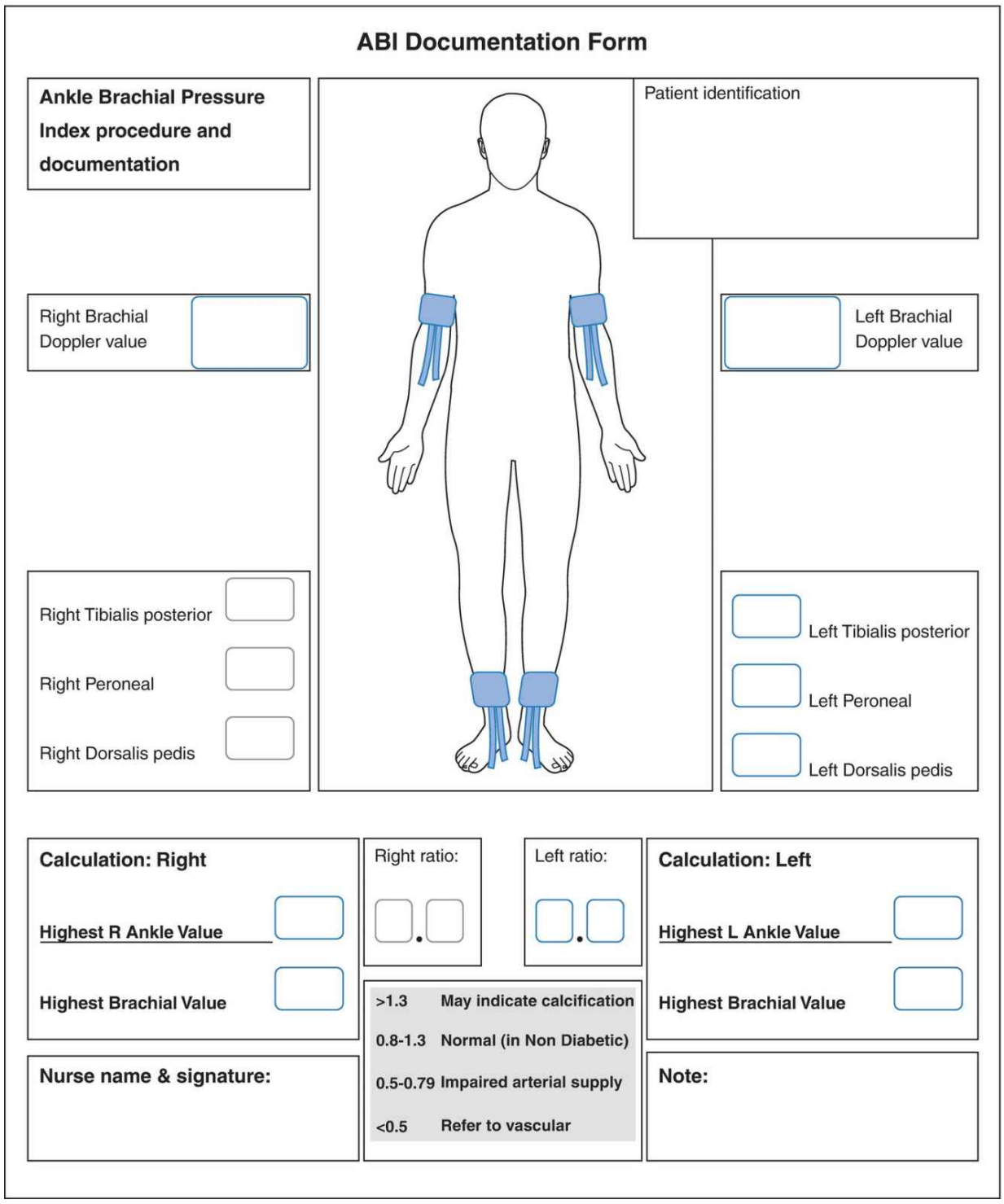

Figure 15-10. Ankle Brachial Pressure Index Procedure and Documentation. (Used with permission H. Smart.)

Physical Examination

Physical examination provides guidance for intervention, starting with skin inspection. Skin changes in venous disease include hyperpigmentation, dermatitis, lipodermatosclerosis, or atrophie blanche. Atrophie blanche is a characteristic white fibrotic scarring at the site of previous ulcers or scattered in patches over the leg with a white indurated skin appearance (Fig. 15-11). These small lesions are mostly deprived of adequate blood supply due to small vessel thrombosis, extremely painful when ulcerated and hard to heal due to additional pain with compression.[35] See Table 15-2 for a comparison of atrophie blanche and livedoid vasculopathy. Because skin color may indicate venous congestion, the color of each toe should be noted and compared with the color of other foot and toes. In venous insufficiency, the skin appears a dusky–ruddy color. In CVI, the skin may become atrophied with scarring from a previous ulcer, or it may have weeping blisters or dry, scaly crusts.

Figure 15-11. Atrophie blanche. (Courtesy R.G. Sibbald, MD.)

Table 15-2 Comparison of Atrophie Blanche Associated with Healed Venous Ulcer with Atrophie Blanche Associated with Livedoid Vasculopathy

Characteristics	AB Associated with Healed Venous Ulcer	AB Associated with Livedoid Vasculopathy
Location	Most commonly on medial ankle	Medial and lateral ankle Extending to dorsum of foot
History	Usually follows an ulcer	May precede, occur at the same time, or follow an ulcer
Morphology	White atrophic scar with areas of megacapillaries	White atrophic scar with areas of megacapillaries plus livedo reticularis
Coagulopathy	Occasional	More common (up to 50%)
Associations	Varicose veins Lipodermatosclerosis	Has two forms of primary and secondary (the secondary form is associated with connective tissue disease, hematologic disorders, and paraproteinemia)
Management	Compression therapy Medical intervention including pentoxifylline Surgical intervention	Anticoagulants Antifibrinolytics Immunosuppressant Immunomodulators Vasodilators Compression therapy

From Alavi, A., Hafner, J., Dutz, JP., et al. Atrophie Blanche: Is It Associated with Venous Disease or Livedoid Vasculopathy? Advances in Skin & Wound Care 27(11):518-24, 2014.

Skin should be palpated for temperature changes or assessed with an infrared no-touch surface thermometer to detect significant temperature differences (3°F or more to depict deep or surrounding wound infection in cases of ulceration.[36] The skin over varicose veins is often warmer than the surrounding skin. Patients presenting with foot or leg ulcers should be tested for neuropathy. This is a common finding in the patient with diabetes.

Edema is commonly found in lower extremity venous disease. Early edema may be observed as a difference in calf circumference between legs and should be confirmed by measurement. After edema

has been long-standing, tissue fibrosis occurs. This makes the skin and subcutaneous tissues less resilient to palpation.

Venous Signs and Symptoms

Patients may report a gradual onset of discomfort associated with venous disease; however, often, no symptoms are present initially. Most patients describe general nondescript aching rather than specific pain. Some terms used to describe sensations in the legs include fullness, swelling, tightness, aching, or heaviness. These symptoms can be reduced with leg elevation. Venous insufficiency accompanied by acute DVT may be described as sharp, severe, deep, aching pain.[37] Varicose veins occasionally produce a pulling, prickling, or tingling discomfort localized to the area of the varicose vein.[7] In severe cases of venous insufficiency, a form of claudication can occur. The patient may complain of foot edema that makes it difficult to wear shoes.

A venous ulcer is moist and may have a yellow fibrous film covering its surface (Fig. 15-12). This fibrous tissue isn't a sign of infection and doesn't interfere with healing.[38] The ulcer edges may be irregular with firm fibrotic and indurated surrounding skin. The surrounding tissue may have a brownish rust color due to erythrocyte breakdown and deposition of hemosiderin. Scar tissue may indicate the site of a previous ulcer. Because of the subcutaneous scarring, there is no allowance for the tissue expansion that occurs with edema in skin of normal elasticity. Scar tissue also prevents blood vessels from transporting oxygen to the skin, further compromising healing. Lipodermatosclerosis is the term used for chronic inflammation and fibrosis of skin and subcutaneous tissues. It may be associated with diffuse inflammatory painful skin edema (see Fig. 15-8).

Figure 15-12. Venous insufficiency ulcer with typical features. (Courtesy J.A. Niezgoda.)

Vascular Testing

Although an experienced vascular clinician can make a vascular diagnosis based on history and physical examination alone, vascular laboratory studies contribute to the accuracy of the diagnosis. The presence, location, and severity of arterial and venous disease are confirmed by vascular laboratory procedures. Information obtained by vascular studies can predict ulcer healing when the cause is arterial insufficiency.[7] Laboratory tests differentiate among conditions that contribute to a nonhealing ulcer.

Noninvasive vascular testing is divided into direct tests that image the vessel itself and indirect tests that demonstrate changes distal to the diseased vessel. These tests range from bedside tests that include Doppler ultrasound to detect the signals of the arteries in the foot with an 8 MHz handheld Doppler probe and determination of an ABI to more complex[39] venous duplex ultrasound, transcutaneous pressure of oxygen ($TCPO_2$), segmental systolic pressures, and plethysmography.

Doppler Ultrasound

In a Doppler ultrasound, a transmitting probe, held at a 45 degree angle sends a signal that is reflected from an object to the receiving probe (usually 8 MHz for vascular assessment). If the signal strikes a moving object such as blood cells, a frequency shift is detected and reflected as sound. The audible signals of venous and arterial flow patterns can be distinguished.

Duplex Ultrasound

The venous duplex allows one to evaluate various segments of the venous tree looking for more than 0.5 seconds reflux after either a muscle contraction or manual augmentation of cephalad flow.[40] An advantage of venous duplex imaging is that it can identify sites of thrombosis with high levels of accuracy. The findings of venous reflex may influence the treatment choice. The disadvantage of this test is that it's fairly time-consuming, taking 1 to 2 hours per evaluation for a full leg imaging.

Venous Testing

It is also possible to use a Doppler probe to perform a crude venous assessment. By compressing the limb manually, the flow in the veins can be augmented and noted by an audible Doppler swishing sound heard distal to the site of compression. This is a subjective test, and reliability is clinician dependent. The introduction of noninvasive vascular testing has provided much anatomical and physiological information to increase the accuracy of diagnosing venous diseases. Two tests have been commonly used to assess the severity of venous insufficiency. One is venous photo plethysmography (PPG), and the other is venous duplex imaging.

Plethysmography

Plethysmography records volume changes in the limb. Several types of plethysmography are available:

- air plethysmography, which uses a pneumatic cuff as a segmental volume sensor
- strain–gauge plethysmography, which uses a fine-bore silicone rubber tube filled with mercury wrapped around the limb to be studied
- impedance plethysmography, which measures the relative change in resistive impedance of the passage of an electrical current through a segment of the body
- PPG, which measures the degree of light attenuation, which is proportional to the quantity of blood present and not actual volume change.[7,40,41]

Air plethysmography and PPG are the more common tests for chronic venous disease. A photoplethysmograph consists of an infrared light-emitting diode and a photo sensor mounted on a probe. The probe is applied to the skin over the area to be tested.[7] The advantage of venous PPG is that it's quick and gives assessment of overall venous refill time. On the other hand, it only evaluates the most dependent portion of the leg in the gator area.

Treating Venous Ulcers

Treatment of venous leg ulcers necessarily involves treatment of the underlying venous disorder. Edema reduction using compression, leg elevation on rest, and an active exercise regimen are components of the ongoing intervention for venous disease.

Treatment goals for all ulcers are to:

- provide an environment conducive to new tissue growth
- protect the wound
- prevent further tissue destruction.

Topical and systemic treatments are addressed simultaneously. It's imperative to consider the cause when deciding treatment because ulcers are not all alike and treatment for one type may be inappropriate or harmful for another type. A vascular specialist consultation is appropriate for ulcers of mixed etiology.

Wound Infection

Infected leg ulcers, soft tissue cellulitis, and osteomyelitis are treated by administering systemic IV or oral antibiotics. Topical antibiotics are not indicated for leg ulcers.[42] Chronic wounds that are colonized with normal skin flora would not be treated with systemic antibiotics if there are no clinical indications of wound or surrounding tissue infection. Rigorous and frequent ulcer cleansing is no longer advised to remove surface bacteria as it adds to wound-related pain that may persist for a prolonged period of time after dressing changes.[42–44] Silver-impregnated dressings have not been shown to have a significant difference in ulcer healing, and routine use of these dressings is not recommended.[38]

Biopsy for a quantitative culture of the inflamed tissue surrounding nonhealing ulcers should be considered if the wound does not progress at least 30% smaller in 4 weeks, remains nonhealed after 12 weeks, or the deep wound infection isn't responding to antibiotics. If carcinoma is suspected, biopsy of the lesion should be obtained (see Chapter 7, Wound Bioburden and Infection).

Visible bone in a wound base or a cavity that can be probed to bone is widely studied in association with diabetic foot osteomyelitis.[45] It should be noted here that probing a wound blindly with anything other than a finger requires an appreciation for the individual's anatomy so as to avoid injuring tissues and blood vessels. Bone scans are expensive and may give false-positive results in the presence of inflammation; therefore, a triple-phase bone scan is indicated only when osteomyelitis is suspected.

Skin and Wound Care

A clean wound, free from dead tissue and wound debris, is necessary for healing to occur. Wound cleaning and debridement are the initial steps in wound care. Many commercial wound cleaners and disinfectants are cytotoxic. Povidone–iodine, hydrogen peroxide, and 0.25% acetic acid have shown evidence of interfering with fibroblast formation and epithelial growth.[46–50] There may be indications for using cidal agents in a wound; however, their use should be time limited, and each caregiver should have a clear understanding of what the goals are and how to recognize when the goals have been reached.

The advantage of cleaning the wound should be weighed against the risk of damaging new tissue growth.[50] The safest wound cleanser is 0.9% saline solution. Wounds should be cleaned with a force strong enough to dislodge debris but gentle enough to prevent damage to newly growing tissue. The pressure to accomplish this goal ranges from 4 to 15 psi.[51] A 19-gauge needle or 19-gauge angiocatheter distributes approximately 8 psi when used with a 35-mL syringe. Commercial irrigation products with caps designed to deliver adequate pressure are available. Leg ulcers treated in the home are commonly irrigated with running tap water, provided the tap water is safe.

Hydrotherapy or whirlpool has been used to aid in cleaning and debridement of both arterial and venous leg ulcers.[52] A clinical pilot study found that whirlpool followed by vigorous rinsing reduced the bacterial load in venous ulcers more than the whirlpool alone.[53] This may suggest that the vigorous irrigation is the significant factor in cleaning the wound. Whirlpool may be contraindicated if the water and dependent position increase edema in the leg. There are no current studies to support or refute whirlpool as a standard recommendation for cleansing venous ulcers.

Dressings chosen for specific wounds depend on the condition of the wound bed and the goal for the wound. Many new dressings are designed to support moist wound healing (see Chapter 9, Wound Care &Treatment Options). Because the skin is fragile in patients with

either arterial or venous disease and can be easily injured, tape and adhesive products should be avoided or used with extreme caution. Use other methods of securing dressings that won't injure the skin. A *Cochrane* review on leg ulcer dressings found that the type of dressing material placed under compression had no influence on healing rates.[54,55] Other studies found no advantages with the use of either silver-impregnated[56] or honey-impregnated[57] dressings. The preferred attachment device for dressings on vascular leg ulcers is roller gauze or commercial devices (such as netting or tube gauze) that hold dressings in place without adhesive, which can damage fragile skin.

Compression

Venous hypertension and venous wounds are treated simultaneously. Wound care and edema management depend on whether or not the patient can be immobilized. Edema is controlled by conservative means: intermittent elevation, compression bandages, and intermittent pneumatic compression.[58] Studies have demonstrated that moist wound healing combined with compression improves wound healing rate of venous ulcers.[59] Compression therapy is the mainstay of venous ulcer therapy. When a person is resting, elevating the legs above the heart is recommended. The current recommendation is to have the individual active and ambulating as soon as possible.[60] Depending on the compression system that is used, patients may be able to remove the compression garment (usually this refers to compression stockings) at night and reapply the garment(s) the next day. If compression bandages are used, they remain in place until they are changed, which is usually weekly.

Compression is the application of pressure to the limb. It is measured in millimeters of mercury (mm Hg) and is applied by bandages, elastic stockings, and/or intermittent pneumatic compression pumps. The amount of compression prescribed is determined by the diagnosis, comorbid conditions, and the patient's ability or willingness to accept the treatment. The following standard pressure classification[61] is suggested: mild (<20 mm Hg), moderate (21 to 40 mm Hg), strong (41 to 60 mm Hg), or very strong (≥61 mm Hg). Compression strength of 30 to 40 mm Hg is recommended to counteract the capillary filling pressures within the leg; however, elderly or frail individuals or those who have difficulty donning the stockings may have compression ordered at 20 to 30 mm Hg. Many factors affect bandage pressure, including the bandage itself, the calf muscle and foot pump function, the shape of the leg, and the skill of the person applying the bandage.

The ambulatory venous patient is best served by semirigid dressings, such as the Unna boot or by multicomponent system compression wraps. Multicomponent compression has been shown to be more effective than single-component compression; both four-layer

and short-stretch bandages have higher healing rates than paste plus an outer support (Unna boot). With the discovery of moist wound healing[62] and the advent of hydrocolloid and foam dressings, occlusive dressings may be used under compression wraps to promote growth of granulation tissue, reduce pain from the dressing rubbing against the ulcer, and promote autolytic debridement. One study found that ulcers treated with the foam dressing under the Unna boot healed twice as fast as ulcers treated without the foam.[63]

Bandages may be made of different materials, including elastic and inelastic materials or both. Stiff bandages are made of multiple layers of elastic or inelastic material. This type of bandage remains rigid and generates high pressure during exercise, which reduces venous hypertension.

Elastic bandages are considered long stretch if they are capable of stretching to double their length and provide sustained pressure even at rest. Because these dressings can be stretched too tight, they are not recommended as a single primary dressing for compression and should be applied within a multiple layer system or method.[64]

Inelastic bandages are nonstretch bandages, short-stretch bandages, and paste bandages. The resting pressure under inelastic bandages decreases over a 24-hour period, but the working pressure (with muscle movement) remains with highest pressure provided when the calf muscle is compressed against the rigid bandage as a fixed resistance when walking. The choice of compression system depends on patient adherence to treatment and clinician competence in application.[65]

Compression wraps should be applied starting just proximal to the toes and ending just below (two finger breadths) the popliteal fossa. A dressing is typically placed over the wound area, and the dressing is covered with a compression bandage system. If the leg is misshapen, padding the leg to create symmetry facilitates a better bandage fit. This will reduce bandage slippage and avoids a tourniquet effect that increases the distal edema. Extra padding around bony prominences reduces the possibility of the compression garment creating a device-related pressure injury. The concave area around the lateral and medial malleoli may need extra padding to ensure sufficient pressure

is applied to those areas. Training should be ongoing to avoid complications associated with the application of compression wraps, such as ineffective pressure, pressure injuries, bandage slippage, and limb distortion[64] (Fig. 15-13).

Figure 15-13. Compression wrapping. This photograph shows a leg being wrapped with a four-layer compression therapy dressing. (Photo courtesy of J.A. Niezgoda.)

Complications from compression include pain, damage to skin and subcutaneous tissue from pressure, reduction of calf muscle from

nonuse, and skin problems. Interventions include carefully assessing to determine the cause of pain; avoiding excessive pressure under the dressing; encouraging exercise, including range of motion; and avoiding topical products that might cause allergies or irritations.[64]

Compression stockings reduce AVP by decreasing venous reflux and improving calf muscle ejection capacity during use.[66] The benefit derived from stockings is in direct proportion to the fit, and there is evidence to support its use as an active venous ulcer treatment.[67]

In many cases, patients fail to wear the prescribed compression dressing or stocking because of difficulty donning the stockings or complaints of tightness. The importance of long-term external compression can't be overemphasized. Patients should be taught that the stockings must be replaced every 3 to 6 months. Two pairs should be purchased so that one can be worn, while the other is laundered.

Long-term compression therapy is an essential part of the treatment of venous leg ulcers to provide a sustained external support to the failed venous valve structures in the leg and is to be maintained for life in patients with lymphedema and venous disease.[62]

A pneumatic compression pump may be used to reduce lower extremity edema. One study found improved venous ulcer healing when compression pumps were used; however, not all third-party payers agree with the use of these pumps for treatment.[68–71] Another study[72] found 4 hours of pump compression per day improved ulcer healing when used in conjunction with compression stockings. In yet another study,[73] an intermittent pneumatic compression device (PCD) provided improved healing when used for 1 hour twice weekly in conjunction with conventional dressings. Patients who are immobile, are unable to tolerate bandages, have arterial insufficiency, or have problems with edema may benefit from intermittent pneumatic compression.[73]

Assessment of Treatment

If a venous ulcer does not show signs of healing, the patient should be reassessed, and a wound edge biopsy is indicated to rule out neoplasms.[74] Concomitant diseases such as diabetes, heart failure, arterial insufficiency, venous reflux, or history of venous thrombosis, end-stage renal disease, critical colonization, and untreated deep wound infection can all lead to slower healing.

Exercise

Evidence supports a graded exercise program as an adjunctive treatment modality in venous leg ulcer care. It improves the calf muscle pump in those patients with abnormalities in pump function and optimizes compression therapy with the calf against a fixed resistance.[60] One author[73] determined that a structured exercise program to improve muscle function may have a significant positive outcome in patients with venous disease.[75]

Effective edema reduction from the leg reverses much of the associated comorbidity, particularly the skin changes. The achieved limb reduction must be maintained through the use of appropriately fitted compression hosiery or an alternative compression product. The application and removal of such hosiery can be problematic for individuals with limited hand function or who are unable to see or reach their toes (obese, blind, arthritic, etc.). However, application aids are available and instruction in technique does help. Other strap-type leggings and boot-like devices are available to decrease the functional burden. Therapists can be very creative in combining or layering different compressive products to achieve adequate levels of compression. Compliance is crucial to prevent recurrence of edema.

Surgical Treatment for Venous Ulcers

Surgical treatment for venous ulcers is aimed at correcting the cause of the venous hypertension. Patients can have venous reflux without the symptoms of insufficiency. It is when reflux is severe enough that the insufficiency results in dermal venous hypertension and the eventual skin changes with which patients present. Procedures aimed at correcting insufficiency of the deep venous system include vein valve transplantation, direct valve repair, and venovenous bypass. Outflow obstruction of a limb is addressed with venovenous bypass, endovascular intervention, or a combination of the two. Varicose veins, the manifestation of superficial venous insufficiency, generally require ablation. Their treatment is usually by excision, ligation, injection, or the more recent method of endovenous ablation, depending on the size of the vein (Fig. 15-14).

Figure 15-14. Ablation of GSV. (Courtesy J. Niezgoda.)

Surgical treatment for venous insufficiency remains far behind the more established treatment of arterial occlusive disease. Venous insufficiency can be grouped under two broad categories:

- venous reflux
- venous outflow obstruction.

The net result of both of these disease entities is venous hypertension and the sequelae resulting in dermal injury that ultimately ends with a venous ulcer. The mainstay for the treatment of venous insufficiency continues to be good external compression. In many patients, this is all that is required. Compression acts both as treatment for various states of venous insufficiency as well as prophylaxis for the development of the adverse sequelae. In some patients, the use of compression alone is inadequate; for these patients, surgical intervention may be necessary.[76]

Venous outflow obstruction is usually the result of DVT; however, other causes include obstruction from surgical excision of outflow vessels, tumor encasement of venous return, radiation-induced fibrosis, congenital atresia, injection-induced venous destruction (illicit or otherwise), obesity with immobility, and infection. When it involves isolated segments with normal segments proximal and distal, the obstruction can result in a cascading event resulting in venous insufficiency. In some patients, the outflow obstruction is the cause of the symptoms. In these patients, it may be necessary to relieve the venous outflow obstruction and the corresponding venous hypertension by bypassing the obstructed segment, by balloon angioplasty of a sclerotic or stenotic segment with or without stent placement, or, if the DVT is not chronic, by either open venous thrombectomy or mechanical thrombectomy.

Endovascular treatment of iliac vein obstruction is now possible on a routine basis. The morphology of this outflow obstruction and the measures to best treat it are ideally evaluated with the use of intravascular ultrasound (IVUS).[77] Although there is an early

reocclusion rate of around 35% at 3 months, subsequent ulcer recurrence-free rates of almost 60% exist out to 5 years.[78] Early thrombosis and in-stent stenosis remain the most common causes of these early failures.

For years, obstruction from DVT has been approached in Europe with open venous thrombectomy, but this technique has never caught on in the United States. Mechanical thrombectomy via percutaneous routes is a focus of interest. The main percutaneous thrombolytic system currently popular is a dispersion and thrombectomy catheter (Bacchus, Santa Clara, CA)[79] and the AngioJet (Possis Medical, Minneapolis, MN).[80,81] This is a unique method of clot destruction and aspiration combined with lytic therapy. The AngioJet uses lytics and a high-velocity jet of liquid to fragment and then rapidly aspirates the thrombus fragments back into the catheter using rheolysis and thrombolysis. The AngioJet also allows pulse-spray thrombolysis to pressure infuse the thrombus with thrombolytic agents. The AngioJet can be used during extracorporal bypass for up to 6 hours and for removal of fresh, soft thrombi. Both methods are effective. Whether these devices will have good long-term results is still in debate. Posttreatment patients need to be on anticoagulation medication.

Patients who require venous bypass are best served using an autogenous venous conduit. The proximal and distal anastomoses of the venous bypass are dictated by the obstruction site. For example, if a patient has an isolated iliofemoral thrombosis that has failed to recannulize or has only partially recannulized and the affected leg is symptomatic, a femoral–femoral bypass from the proximal portion of the symptomatic leg to the more distal portion of the contralateral leg can be performed. The saphenous vein is usually used for this, but in contrast to arterial bypasses, the direction of the valve isn't reversed; rather the valve leaflets are oriented to prevent reflux. Similarly, a bypass from a more proximal vein within a symptomatic leg to the more cephalad iliac vein may be indicated.[82]

In patients with an outflow obstruction, but in whom insufficiency or hypertension is caused by occlusion of the GSV, the venous hypertension may be alleviated by isolated partial saphenous vein ligation and stripping. This is usually done at the knee level with

stripping of the affected saphenous segment. If, however, the reflux or hypertension is the result of the deep venous system, then stripping the GSV wouldn't help and actually may be detrimental due to elimination of one of the venous outflow tracts of the extremity. This information must be known before a surgical procedure is performed to correct venous insufficiency. Three tests are available to evaluate reflux:

- ascending/descending venography
- duplex imaging
- venous PPG with sequential tourniquet placement.

These tests are all readily available through either an accredited vascular laboratory or a radiology department with experience in venous testing. Venous PPG can determine whether the deep or superficial system is involved with venous reflux. It is an excellent assessment of overall limb reflux and helps assess for perforator incompetence. Venous duplex, which interrogates specific segments of both the superficial and deep veins for reflux, gives a more detailed anatomic evaluation of the affected extremity. Both are needed to properly evaluate a patient with venous disease. Isolated valve segments of the more proximal venous system can be evaluated using descending venography looking for contrast reflux past the incompetent valve. Venography and venous duplex imaging can also determine sites of stenoses within the venous system. One of the problems associated with accurate duplex imaging is that it's dependent upon the competence of the technician performing the test. Certain laboratories have more expertise in these areas than others do. This notwithstanding, duplex imaging has largely replaced venography in evaluating patients with venous disease. Venography may still be used by some clinicians if surgical bypass and/or valvular surgery is planned.

When deep venous insufficiency is due to valvular incompetence, it isn't known how many competent valves are required and in what locations for the deep venous system to become competent again. Research in these areas is still ongoing as more attention is given to venous insufficiency.[82]

Three techniques are available for surgical correction of venous insufficiency due to valvular incompetence:

- artificial venous valve insertion
- autogenous vein valve transplantation using a segment of vein, usually from the upper extremities or axilla
- direct valvuloplasty.

Autogenous valve transplantation is a procedure in which a segment of vein with a competent valve, usually from the upper extremity or axilla, is identified. This section of vein is resected and an interposition graft placed at the harvest site. The vein is then transposed into the venous system of the affected extremity, maintaining the orientation of the valve to keep the leaflets open in a cephalic direction. Postoperative anticoagulation is commonly used.

Approximately 75% of the venous ulcers remain healed 12 months after valve transplantation.[83] However, considerable degradation occurred over the course of the 2nd year in these patients, such that only 40% of limbs remain healed.[84] After the 2nd year, results appear to stabilize without further deterioration although the reports on this are limited in both scope and number.[82]

Variations of this procedure using valve segments from other areas or even transposing a deep vein with a competent valve with another deep vein with an incompetent valve can be done. In one study, the overall results with this method appeared to be similar to that of transposition of competent vein valve segments.[84]

Direct valvuloplasty is another technique for correcting valvular insufficiency. This is performed by suture approximation of two valve leaflets that don’t fully close. It’s done either with direct suturing within the areas of the cusps to obtain good apposition or by external buttressing of an incompetent vein valve sinus. Valve leaflets themselves are brought into apposition by placing the equivalent of a “girdle” around the dilated valve to reduce dilation and allow the valves to come in apposition in a more normal fashion. This sleeve technique is usually done with prosthetic material. Similarly, a transplanted valve that may deteriorate due to dilation can be made competent again by using this technique.[83]

An external valve repair known as the *Psathakis silastic sling procedure* has been developed.[84] This procedure involves placing a silastic sling around the popliteal vein and then attaching it to the two heads of the biceps femoris muscle. When these muscles contract, the sling is intended to occlude the popliteal vein during ambulation. The problem with this procedure is that the sling becomes intimately adherent to the vein and surrounding tissue and over time no longer functions in this fashion.

Two publications on neovalve construction and valvular repair highlight the various techniques employed to restore venous competency of the deep system and their outcomes.[85,86] These are technically challenging operations that are not widely available. When successful, ulcer healing rates exceed 88%.

In patients in whom no suitable vein valve segment can be found or it's deemed an inadequate operation, the development and implantation of a prosthetic valve holds some promise. Currently, a prosthetic venous valve comprised of a complex titanium double-leaflet system is being developed and may hold promise.

The appropriate use of adequate compression is necessary in conjunction with all the surgical treatments. The application of compression hose and management of these patients are addressed elsewhere within this chapter.

Historically, patients with recurrent leg ulcers due to incompetent perforators in the affected area were thought to benefit from a Linton flap. This procedure requires elevation of the skin and fascia at the site of ulceration and transection with ligation of the incompetent perforator veins feeding the area. Proper application of compression is required afterward to reduce local venous hypertension. The morbidity associated with this procedure includes tissue slough along the area of incision and the overlying tissue, resulting in a prolonged healing period. This is thought to be due to the chronic disease state of the tissue at the ankle.

A subfascial ligation of incompetent perforator veins with an endoscope (subfascial endoscopic perforator surgery [SEPS]) was thought to be a significant advancement in the Linton technique. Using equipment developed for laparoscopic cholecystectomies, the scope

is passed from the medial infrageniculate area into the subfascial space. The fascia is raised, often with CO_2 insufflation. The perforator veins are ligated and transected through the endoscope. Advances in technology, such as smaller-diameter scopes (5 to 10 mm), high-resolution camera and LCD screens, and single-step electrocautery/transection, have made this procedure much easier to perform. The smaller-diameter endoscopes allow easy perimalleolar inspection for perforators. This entire procedure routinely takes less than an hour to perform and in select patients can be done on an outpatient basis. The reason for ligating incompetent perforators is to eliminate the venous hypertension associated with the reflux of venous blood.[87] However, the contribution of incompetent perforators to venous insufficiency remains controversial.[88]

In a meta-analysis by Tenbrook et al., ulcers treated by SEPS with or without additional venous ablation healed in 88% of patients.[89,90]

There has been some interest in duplex ultrasound–guided foam sclerotherapy, which scleroses the perforator veins to achieve the same effect.[91,92] While this treatment is used extensively in Europe, it's not yet approved in the United States.[93] Further studies are needed to establish the overall effectiveness of this procedure for wound healing.

Foam Sclerotherapy

Chemical agents, when injected into veins, cause luminal narrowing and eventual occlusion secondary to intimal damage and inflammation. This obliterates the vein. Veins with faulty valves and varicosities can be treated this way. Foaming the sclerosant leads to increase in the agent's surface area, diminishes its propagation, and allows a better therapy result than using liquid sclerosant. Foam sclerotherapy can be performed in a clinical outpatient setting. It may potentially require local anesthetic under ultrasound guidance. Following the treatment, it is important for the patient to wear a compression bandage or support stocking on the leg for a designated period of time. Success rates are approximately 70% to 80%. If the veins do not disappear, the treatment can be repeated. Side effects may be due to reactions to the foam entering the general circulation. They include visual disturbances which usually are transient. Local "phlebitis" of the treated vein may occur in veins that are superficial. There may be local pain for 1 to 2 weeks and may occasionally leave a dark linear mark across the skin. Elevation, warm compresses, and anti-inflammatory medications are used for symptom management.[90]

The foam can be produced manually just prior to injection using the Tessari (@) method. Foam prepared according to the Tessari technique has a half-life of approximately 90 seconds. Thus the procedure should be completed within 1 minute, before the foam separates. Phase III trials (VANISH-1 and VANISH-2)[93] have confirmed the safety and efficacy of polidocanol endovenous microfoam (Varisolve) for improving symptoms when used to treat superficial venous incompetence, leading to approval in the United States in 2013.

Sclerosant foam is echogenic under ultrasound, secondary to the air within the foam. The foam, with its increased surface area, is much more effective than liquid sclerosants. The foam also allows for a lower volume of medication to be injected. In principle, all vein calibers are suitable for foam sclerotherapy. This includes the tributaries of the saphenous system, the reticular varicosities, and the telangiectasias. Ultrasound-guided foam sclerotherapy (UGES) is predominantly

applied to the treatment of saphenous tributaries and incompetent perforating veins.

Sclerotherapy Agents

The most common sclerosing agents used in the treatment of lower extremity telangiectasias, reticular veins, and small varicose veins are sodium tetradecyl sulfate, polidocanol, glycerin, and hypertonic saline. These substances cause endothelial damage by either osmotic or detergent agents. Osmotic agents are effective by dehydrating endothelial cells. Detergents damage the endothelium by interfering with cell membrane lipids.

Sodium tetradecyl sulfate (Sotradecol): This is the most commonly studied sclerosant. The maximum recommended dosage is 10 mL of a 3% solution in the United States and Canada. The volume used varies worldwide depending on formulation. Dilutions between 0.1% and 3.0% are used depending upon the size of the vein treated.

Polidocanol: This is also known as aethoxysklerol. It is a detergent. It is now available in the United States for commercial use under the name Asciera.

Hypertonic saline: This is the most commonly used osmotic agent for lower extremity sclerotherapy. The dilution used to treat lower extremity vessels ranges from 11.7% to 23.4%.

Glycerin: Chromated glycerin (e.g., Chromex Skermo) is a potent osmotic with dilutions that range from 25% to 72%. The maximum recommended amount per session is 10 mL. While popular worldwide, it is not commercially available in the United States.

Mechanical–chemical vein ablation: This is a mechanism to deliver a chemical sclerosant directly into the saphenous trunk via a catheter that spins and agitates, thereby damaging the intimal lining of the vein. ClariVein is an example of this technology. The potential advantage of this modality is that it is less uncomfortable and requires less local anesthetic than the thermal techniques. The long-term durability of this technique is still being evaluated.

Cyanoacrylate glue: Glue was initially described for treatment of saphenous incompetence in 2013.[94] The cyanoacrylate has been used in the treatment of arteriovenous malformations for over 30 years. One of the advantages of this treatment is the lack of need for

tumescent anesthesia which can be a patient deterrent and hindrance in the heat-induced techniques. In a randomized trial comparing this system with radiofrequency ablation, short-term outcomes at 3 months were similar. Long-term follow-up is needed to determine the durability of the results.[95]

The agent is injected sequentially through a catheter followed by compression along the length of the vein from proximal to distal. If the first injection is approximately 5 cm from the saphenofemoral junction, the vein above the catheter is compressed with the ultrasound probe prior to the injection to prevent injecting into the deep venous system. The feasibility of the use of this system for perforating veins was investigated in a separate study. There were no serious complications.[96]

The use of endovenous ablation has become widespread in the United States, supplanting traditional vein stripping. In most places, endovenous ablation is an outpatient office-based procedure. It involves ultrasound-guided cannulation of the distal saphenous vein, either lesser or greater, with a catheter whose tip is positioned 2 cm distal to the saphenofemoral junction. The length of the vein is surrounded with tumescent anesthesia usually consisting of a dilute buffered lidocaine solution (usually 300 to 500 mL). This acts as a heat trap for the delivered energy, which destroys the vein wall and coagulates the blood within. The energy delivered is either in the form of a laser (endovenous laser therapy [EVLT]) or radiofrequency (RF).97 Proponents of both forms claim superiority. The end result, if successful, is controlled thrombosis and destruction of the vein and thereby prevention of reflux through it. US data show 99.6% successful occlusion initially, falling to 86% to 89% at 4 years.[98] Devices of both types exist that can be used to directly ablate incompetent perforators under ultrasound guidance. However, neither of these devices is as easy to use as the main units.

RF and EVLT are quick procedures, routinely taking far less than 1 hour to perform. The patients generally do very well, and the procedures are well tolerated. The patient avoids a hospital stay and the need for a major anesthetic. Endovenous ablation has been widely accepted as a tool for the patient seeking removal of mostly

asymptomatic varicosities, but when appropriately applied, the procedure can correct isolated saphenous reflux disease. While the procedure has a low complication rate, it has two major drawbacks, while open vein stripping does not: it usually leaves behind a distal segment of vein, and recannulation can occur. Some centers have combined endovenous ablation with a subsequent limited distal stripping or microphlebectomy, if needed. Depending on how extensive the condition, either of these procedures could be office based.

In some patients, the application of a split-thickness skin graft to an otherwise healthy venous ulcer may be appropriate. This technique should not be used in patients whose underlying venous problems have not been addressed. The application of a split-thickness skin graft to an ulcer with persistent venous hypertension will fail, even if the patient is compliant with a compression garment.

Options other than split-thickness grafting involve the use of bioengineered human dermal substitutes. These are intended to be used as carriers of human growth stimulants and to provide a scaffold upon which the patient's own dermis can regenerate, either spontaneously or with the use of a delayed thin skin graft. Dermal substitutes are not inexpensive, however, and the underlying venous issue as well as the wound bed must still be addressed. Although skin substitutes appear to hasten wound closure, recurrence rates are high.

Modern venous surgery now has in its' armamentarium a wider array of treatment options for those patients with venous disease. As this area of vascular surgery matures, better paradigms for patient treatment are emerging. No longer is it just an Unna boot and vein stripping.

Summary

CVI is a permanent condition. Because of this, patients are given information about the disease process and rationale for intervention. The more information they have, the more likely they are to manage the condition effectively. Activities that promote venous return are encouraged. Extremity elevation at rest should become a daily routine, and external compression is needed for life. This should be a well-structured patient education piece that may need to be repeated several times to improve patient adherence to treatment and long-term preservation of intact skin in healed ulcers. Protection from skin trauma is essential. A small lesion may progress quickly to a large ulcer because of the edema. It may take years to heal, if at all. Small cuts or bruises should receive immediate medical attention. Leg exercises to increase muscle pump activity are taught to the patient. Patients are encouraged to use these exercises during long periods of standing or sitting. When sitting, the legs should be elevated to the level of the heart or above if possible.

Success in managing venous ulcers requires a total patient commitment. Risk factors and ulcer management are dependent upon the patient's activities; therefore, the patient must have as much information as possible to participate in therapy. An understanding of venous pathophysiology and its contribution to leg ulcers is critical in managing the ulcers. Venous reconstruction is in its infancy but shows promise to reduce the sequelae of postphlebitic syndrome. Venous ulcers always require external compression, ultimately in the form of compression stockings or layered bandaging systems.

A variety of wound care products are available for leg ulcers, but no research exists showing one product to be more effective than another. Economic concerns make it imperative to choose the appropriate dressings and treatment, but research demonstrates little increased benefit of the newer treatments over the old. Although surgical treatment for venous ulcer lags behind that for arterial occlusive disease, there are procedures that correct the insufficiency of the deep venous system. These are vein valve transplantation, direct valve repair, and venovenous bypass.

The mainstay for venous lower limb ulcer care continues to be external compression, either in the form of compression stockings or layered bandaging systems. The importance of clinical competence in application to the benefit of the patient and ultimate healing outcomes cannot be over emphasized. This will ensure compression as a modality to remain the safe, efficient, and cost-effective modality it has proven to be over time.

PATIENT SCENARIO

Clinical Data

Mrs. MC is an 82-year-old woman who has had multiple recurrent superficial ulcers in the medial aspect of the left ankle region, presumably resulting from venous insufficiency. Some ulcers healed spontaneously, but more recently, they required compression therapy at a wound center. The current episode was indolent for more than 8 months and has left Mrs. MC with macerated skin that cracks and weeps serous fluid (Fig. 15-15A). She recently moved in with her daughter but is quite independent. Although Mrs. MC is thin, her nutrition is adequate and she is compliant with leg elevation. She has worn proper compression hose in the past. She was referred to the Vascular Surgery Service for evaluation.

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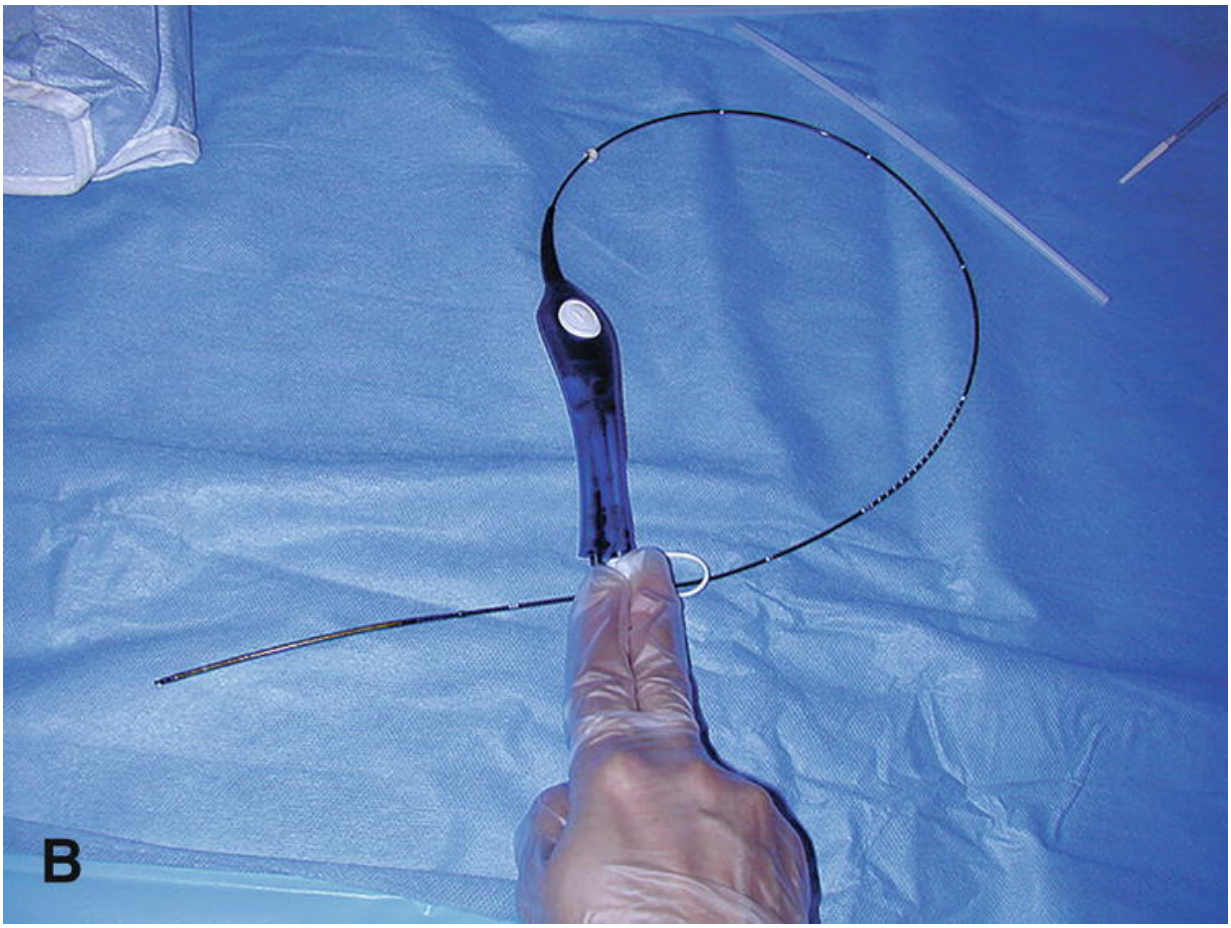
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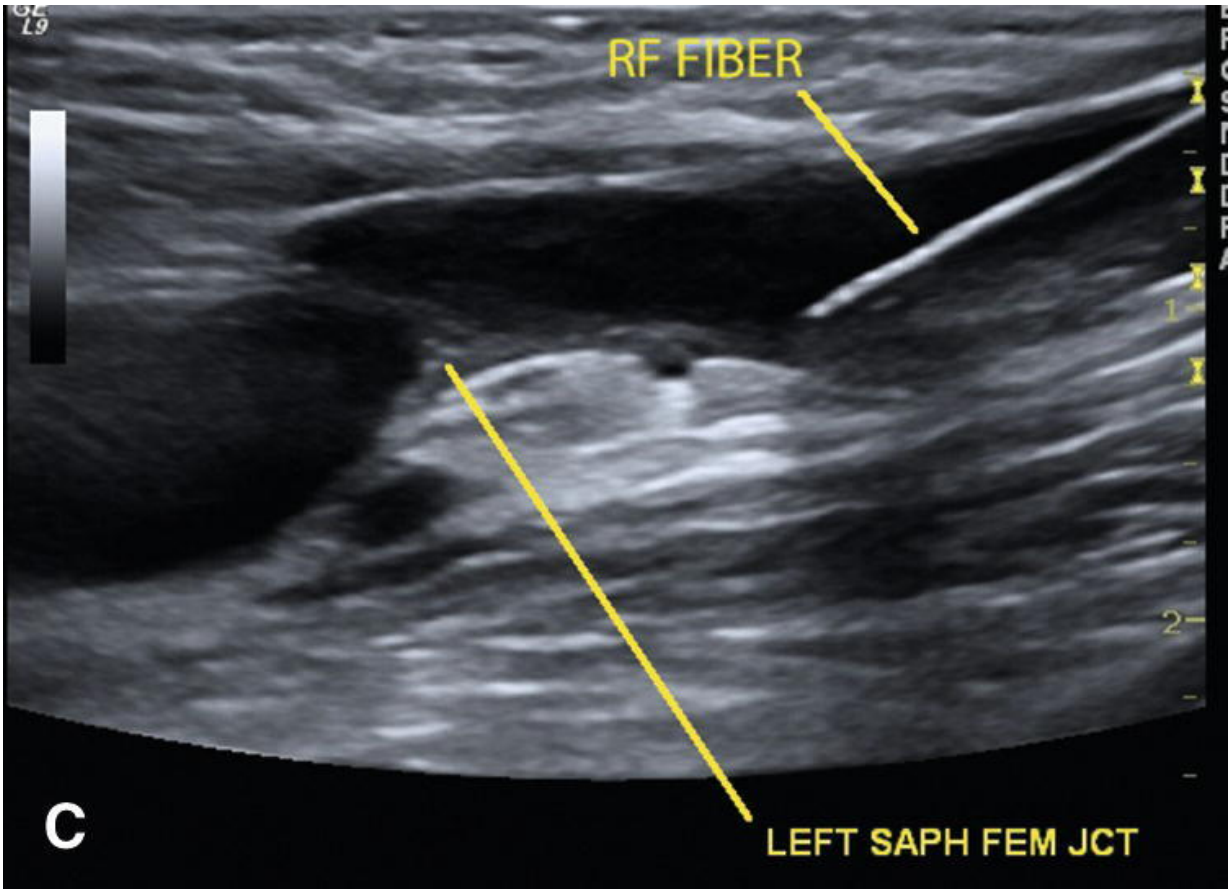
RF FIBER
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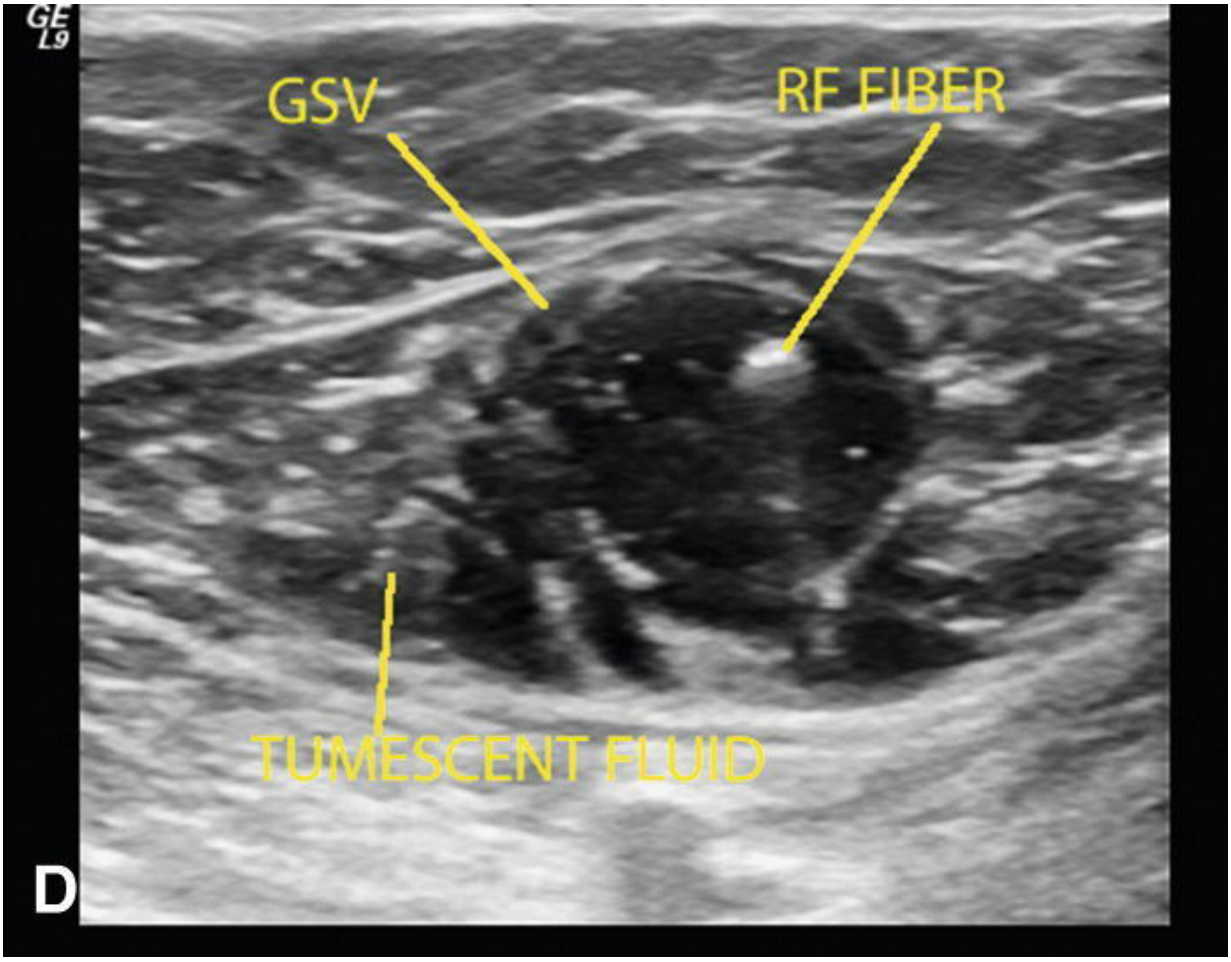

Figure 15-15.

The left leg has no palpable pedal pulses, although Mrs. MC does not describe any claudication or rest pain. She has a remote history of limited smoking. Other than hypertension and the current left leg complaints, she is healthy. She underwent arterial and venous vascular lab evaluations consisting of arterial Doppler ultrasound, venous PPG, and venous duplex tests for reflux. Her ABIs were greater than 0.95 bilaterally with triphasic waveforms. Venous refill times were markedly abnormal bilaterally, 12 seconds on the left and 15 seconds on the right. Tourniquet application indicated isolated GSV incompetence in both legs. Venous duplex testing revealed no DVTs, greater than 0.5 seconds of reflux in the left proximal femoral vein and the entire left GSV. Reflux was also noted in the entire right GSV. Endovenous ablation of the left GSV was recommended.

Case Discussion

Mrs. MC's GSV incompetence on her symptomatic left side is most likely the cause of her recurrent venous ulcers and now chronic tissue and skin changes. Although there is reflux in the left proximal femoral vein, the GSV is the predominant source of her dermal venous hypertension. With ablation of this GSV and, once healed, wearing proper support hose, it is expected that her symptoms will improve and her ulcer will heal. Because of her age and the desire to avoid both hospitalization and a major anesthetic, endovenous ablation with RF was the intervention of choice. This office-based procedure requires only local tumescent anesthesia.

The RF fiber (Fig. 15-15B) was passed up the GSV after entry at the distal calf level via ultrasound guidance. After placement approximately 2 cm distal to the junction of the common femoral vein (Fig. 15-15C), tumescent infusion around the vein was performed (Fig. 15-15D). Several cycles of heat were applied as the fiber was sequentially pulled back. Once completed, the limb was compressed using thigh-high support hose of 30 to 40 mm Hg compression; support hose were worn for an initial 72 continuous hours followed by 7 additional days of nonsleep wear. The patient then returned for a series of duplex ultrasound tests to evaluate the ablation and to rule out DVT.

Mrs. MC is now doing well with well-healed skin. This took an additional 2 months to achieve. She wears knee-high support hose of 30 to 40 mm Hg compression religiously on both limbs. The right leg remains asymptomatic.

Show What You Know: Venous Disease

1. **The cause of venous ulcers is:**
   A. venous stasis.
   B. venous hypertension.
   C. embolic phenomenon.
   D. varicose veins.

2. **The ankle–brachial index (ABI) is an indicator of loss of perfusion in the lower extremity.**
   A. True
   B. False

3. **The most important treatment component for venous ulcers is:**
   A. moist wound healing.
   B. antibiotics.
   C. compression.
   D. revascularization.

4. **The most detrimental activity a patient with any vascular disease can do is:**
   A. walk into the pain.
   B. sleep with legs dependent.
   C. use nicotine.
   D. fail to monitor pulses.

Answers to these questions are found on page 685.

References

1. Martinengo, L., et al. Prevalence of Chronic Wounds in the General Population: Systematic Review and Meta-analysis of Observational Studies. Annals of Epidemiology 29:8e15, 2019.
2. Rice, J.B., et al. Burden of Venous Leg Ulcers in the United States. Journal of Medical Economics 17:347-56, 2014.
3. Coon, W.W., et al. Venous Thromboembolism and Other Venous Disease in the Tecumseh Community Health Study. Circulation 48(4):839-46, October 1973.
4. Dewolfe, V.G. “The Prevention and Management of Chronic Venous Insufficiency.” Practical Cardiology 6:197-202, 1980.
5. Callam, M.J., et al. “Chronic Ulcers of the Leg: Clinical History.” British Medical Journal (Clinical Research Edition) 294(6584):1389-91, May 1987.
6. Nelzen, O., et al. “The Prevalence of Chronic Lower-limb Ulceration has been Underestimated: Results of a Validated Population Questionnaire.” British Journal of Surgery 83(2):255-8, February 1996.
7. Sidway, A.N., Perler, B.A. Rutherford Vascular Surgery and Endovascular Therapy, 9th ed. Philadelphia, PA: WB Saunders, 2018.
8. Moore, W.S., ed. Vascular and Endovascular Surgery: A Comprehensive Review, 8th ed. Philadelphia, PA: Elsevier Saunders, 2013.
9. Browse, N.L., et al. Diseases of the Veins: Pathology, Diagnosis, and Treatment. London, UK: Edward Arnold, 1988.
10. Phillips, T.J., Dover, J.S. “Leg Ulcers.” Journal of the American Academy of Dermatology 25(6 Pt 1):965-89, December 1991.
11. Recek, C. Calf Pump Activity Influencing Venous Hemodynamics in the Lower Extremity. The International Journal of Angiology 22(1):23-30, 2013.
12. Levick, J. “Revision of the Starling Principle: New Views of Tissue Fluid Balance.” Journal of Physiology 557(3):704, 2004.
13. Browse, N.L., Burnand, K.G. “The Cause of Venous Ulceration.” Lancet 2(8292):243-5, July 1982.
14. Sutcliffe, J.E.S., et al. Changes in the Extracellular Matrix Surrounding Human Chronic Wounds Revealed by 2-Photon Imaging. International Wound Journal 14(6):1225-36, 2017.
15. Dodd, H., Cockett, F. “The Postthrombotic Syndrome and Venous Ulceration.” In H. Dodd, F. Cockett, eds. The Pathology and Surgery of the Veins of the Lower Limbs. New York: Churchill Livingstone, 1976.
16. Burnand, K., et al. “Venous Lipodermatosclerosis: Treatment with Fibrinolytic Enhancement and Elastic Compression.” British Medical Journal 280(6206):7-11, January 1980.
17. Powell, S. “Contact Dermatitis in Patients with Chronic Leg Ulcers.” Journal of Tissue Viability 6(4):103-6, October 1996.

18. Owens, J.C. “Management of Postphlebitic Syndrome.” British Journal of Surgery 68:807-96, 1981.
19. Nicolaides, A., et al. “Chronic Deep Venous Insufficiency.” In H. Haimovici, et al., eds. Haimovici’s Vascular Surgery, 4th ed. Oxford: Blackwell Science, 1996.
20. Nicolaides, A.N., et al. Investigation of CVI. A Consensus Statement. Circulation 102(20):E126–63, 2000.
21. Blalock, A. “Oxygen Content of Blood in Patients with Varicose Veins.” Archives of Surgery 19:898-905, 1929.
22. Lindemayr, W., et al. “Arteriovenous Shunts in Primary Varicosis? A Critical Essay.” Vascular Surgery 6(1):9-13, January-February 1972.
23. Burnand, K.G., et al. “Peripapillary Fibrin in the Ulcer-bearing Skin of the Leg: The Cause of Lipodermatosclerosis and Venous Ulceration.” British Medical Journal (Clinical Research Edition) 285(6): 1071-2, November-December 1982.
24. Falanga V., et al. “Pericapillary Fibrin Cuffs in Venous Ulceration: Persistence with Treatment and During Ulcer Healing.” The Journal of Dermatologic Surgery and Oncology 18(5):409-14, May 1992.
25. Coleridge Smith, P.D., et al. “Causes of Venous Ulceration.” British Journal of Hospital Medicine 296 (6638):1726-7, June 1988.
26. Sarin, S., et al. “Disease Mechanisms in Venous Ulceration.” British Journal of Hospital Medicine 45(5): 303-5, May 1991.
27. Falanga, V., Eaglstein, W.H. “The ‘Trap’ Hypothesis of Venous Ulceration.” Lancet 341(8851):1006-8, April 1993.
28. Galsinh H, et al. Lipodermatosclerosis: The Common Skin Condition Often Treated as Cellulitis. The Journal of the Royal College of Physicians of Edinburgh 49(1):41-2, 2019.
29. Porter, J.M., et al. “Reporting Standards in Venous Disease.” Journal of Vascular Surgery 8(2):172-81, August 1988.
30. Ad Hoc Committee of the American Venous Forum. Classification and Grading of Chronic Venous Disease in the Lower Limbs: A Consensus Statement. In P. Gloviczki, J.S.T. Yao, eds. Handbook of Venous Disorders: Guidelines of the American Venous Forum. London, UK: Chapman & Hall, 1996.
31. Kistner, R.L., et al. “Diagnosis of Chronic Venous Disease of the Lower Extremities: The CEAP Classification.” Mayo Clinic Proceedings 71(4):338-45, April 1996.
32. Eklof, B., Rutherford, R.B., Bergan, J.J., et al. Revision of the CEAP Classification for Chronic Venous Disorders: Consensus Statement. Presented at the Sixteenth Annual Meeting of the American Venous Forum, Orlando, FL, February 26-29, 2004.
33. Rutherford, R.B., Padberg, F.T. Jr, Comerota, A.J., et al., “American Venous Forum’s Ad Hoc Committee on Venous Outcomes Assessment. Venous

Severity Scoring: An Adjunct to Venous Outcome Assessment." Journal of Vascular Surgery 31:1307-12, 2000.
34. Vasquez, M.A., Rabe, E., McLafferty, R.B., et al. "Revision of the Venous Clinical Severity Score: Venous Outcomes Consensus Statement: Special Communication of the American Venous Forum Ad Hoc Outcomes Working Group." Journal of Vascular Surgery 52(5):1387-96, 2010.
35. Alavi, A., Hafner, J., Dutz, JP., et al. Atrophie Blanche: Is It Associated with Venous Disease or Livedoid Vasculopathy? Advances in Skin & Wound Care 27(11): 518-24, 2014.
36. Mufti, A., et al. Infrared Skin Thermometry: Validating and Comparing Techniques to Detect Periwound Skin Infection. Advances in Skin & Wound Care 31(1):607-11, 2018.
37. Fahey, V.A., White, S.A. "Physical Assessment of the Vascular System." In V.A. Fahey, eds. Vascular Nursing. Philadelphia, PA: WB Saunders, 1994.
38. O'Donnell, T.F., et al. Management of Venous Leg Ulcers: Clinical Practice Guidelines of the Society for Vascular Surgery and the American Venous Forum. Journal of Vascular Surgery 60:3S-59S, 2014.
39. Alavi, A., et al. "Audible Handheld Doppler Ultrasound Determines Reliable and Inexpensive Exclusion of Significant Peripheral Arterial Disease." Vascular 23(6): 622-9, 2015.
40. Coleridge-Smith, P., et al. "Duplex Ultrasound Investigation of the Veins in Chronic Venous Disease of the Lower Limbs—UIP Consensus Document. Part 1. Basic Principles." European Journal of Vascular Surgery 31:83, 2006.
41. Nicolaides, A.N., Miles, C. "Photoplethysmography in the Assessment of Venous Insufficiency." Journal of Vascular Surgery 5(3):405-12, March 1987.
42. Burton, C. "Venous Ulcers." American Journal of Surgery 167(1A Suppl):S37-S41, January 1994.
43. Woo, K.Y., Sibbald, R.G. "A Cross Sectional Validation Study of Using NERDS and STONES to Assess Bacterial Burden." Ostomy/Wound Management 55(8): 40-8, 2009.
44. Price, P.E., et al. Dressing Related Pain in Patients with Chronic Wounds: An International Patient Perspective. International Wound Journal 5(2):159-71, 2008.
45. Lam, K., et al. Diagnostic Accuracy of Probe to Bone to Detect Osteomyelitis in the Diabetic Foot: A Systematic Review. Clinical Infectious Diseases 63(7):944-8, 2016.
46. Lineaweaver, W., et al. "Topical Antimicrobial Toxicity." Archives of Surgery 120(3):267-70, March 1985.
47. Lineaweaver, W., et al. "Cellular and Bacteriologic Toxicities of Topical Antimicrobials." Plastic and Reconstructive Surgery 75(3):94-6, March 1985.
48. Cooper, M., et al. "The Cytotoxic Effects of Commonly Used Topical Antimicrobial Agents on Human Fibroblasts and Keratinocytes." Journal of Trauma 31(6): 775-84, June 1991.

49. McCauley, R.L., et al. “In Vitro Toxicity of Topical Antimicrobial Agents to Human Fibroblasts.” Journal of Surgical Research 46(3):267-74, March 1989.
50. Maklebust, J. “Using Wound Care Products to Promote a Healing Environment.” Critical Care Nursing Clinics of North America 8(2):141-58, June 1996.
51. Maklebust, J., Sieggreen, M. Pressure Ulcers: Guidelines for Prevention and Management, 3rd ed. Springhouse, PA: Springhouse Corp., 2001.
52. Niederhuber, S.S., et al. “Reduction of Skin Bacterial Load with Use of Therapeutic Whirlpool.” Physical Therapy 55(5):482-6, May 1975.
53. Bohannon, R.W. “Whirlpool versus Whirlpool and Rinse for Removal of Bacteria from a Venous Stasis Ulcer.” Physical Therapy 62(3):304-8, March 1982.
54. O’Meara, S., et al. “Compression for Venous Leg Ulcers.” Cochrane Database of Systematic Reviews 11: CD000265, 2012.
55. Palfryman, S.S.J., et al. “Dressings for Healing Venous Leg Ulcers.” Cochrane Database of Systematic Reviews 3:CD001103, 2006.
56. Michaels, J., et al. “Randomized Controlled Trial and Cost Effectiveness Analysis of Silver-donating Antimicrobial Dressings for Venous Leg Ulcers (VULCAN Trial).” British Journal of Surgery 96:1147-56, 2009.
57. Jull, A., et al. “Randomized Clinical Trial of Honey-impregnated Dressings for Venous Leg Ulcers.” British Journal of Surgery 95:175-82, 2008.
58. Goldman, M.P., et al. “Diagnosis and Treatment of Varicose Veins: A Review.” Journal of the American Academy of Dermatology 31(3 Pt 1):393-416, September 1994.
59. Cordts, P.R., et al. “A Prospective, Randomized Trial of Unna’s Boot Versus Duoderm CGF Hydroactive Dressing Plus Compression in the Management of Venous Leg Ulcers.” Journal of Vascular Surgery 15(3): 480-6, March 1992.
60. Klozizakis, M., et al. “Supervised Exercise Training as an Adjunct Therapy for Venous Leg Ulcers, A Randomized Controlled Feasibility Trial.” The British Journal of Dermatology 178(5):1072-82, 2018.
61. Partsch, H. et al. “Classification of Compression Bandages: Practical Aspects.” Dermatologic Surgery 34:600-9, 2008.
62. Winter, G.D. “Formation of a Scab and the Rate of Epithelialization of Superficial Wounds in the Skin of a Pig.” Nature 193:293-4, 1962.
63. Loiterman, D.A., Byers, P.H. “Effect of a Hydrocellular Polyurethane Dressing on Chronic Venous Ulcer Healing.” Wounds 3(5):178-81, September-October 1991.
64. Health Quality Ontario. Compression Stockings for the Prevention of Venous Leg Ulcer Recurrence: A Health Technology Assessment. Ontario Health Technology Assessment Series 19(2):1-86, 2019.
65. DeCarvalho, M.R., et al. A Meta-analysis to Compare Four Layer to Short Stretch Compression Bandaging for Venous Leg Ulcer Healing. Ostomy/Wound Management 64(5):30-7, 2018.

66. World Union of Wound Healing societies (WUWHS). Principles of Best Practice: Compression in Venous Leg Ulcers. A Consensus Document. London, UK: MEP Ltd, 2008.
67. Rabe, E., et al. Indications for Medical Compression Stockings in Venous and Lymphatic Disorders: An Evidence-Based Consensus Statement. Phlebology 33(3):163-84, 2018.
68. Noyes, L.D., et al. “Hemodynamic Assessment of High Compression Hosiery in Chronic Venous Disease.” Surgery 102(5):813-5, November 1987.
69. Pekanmaki, K., et al. “Intermittent Pneumatic Compression Treatment for Postthrombotic Leg Ulcers.” Clinical and Experimental Dermatology 12(5):350-3, September 1987.
70. Scurr, J.H., et al. “Regimen for Improved Effectiveness of Intermittent Pneumatic Compression in Deep Venous Thrombosis Prophylaxis.” Surgery 102(5): 816-20, November 1987.
71. Mulder, G., et al. “Study of Sequential Compression Therapy in the Treatment of Nonhealing Chronic Venous Ulcers.” Wounds 2:111-5, 1990.
72. Allsup, D.J. “Use of the Intermittent Pneumatic Compression Device in Venous Ulcer Disease.” Journal of Vascular Nursing 12(4):106-11, December 1994.
73. Smith, P.C., et al. “Sequential Gradient Pneumatic Compression Enhances Venous Ulcer Healing: A Randomized Trial.” Surgery 108(5):871-5, November 1990.
74. Margolis, D.J., et al. “Risk Factors Associated with the Failure of a Venous Leg Ulcer to Heal.” Archives of Dermatology 135(8):920-26, 1999.
75. Sibbald, R.G., et al. Special Considerations in Wound Bed Preparation 2011: An Update. Advances in Skin & Wound Care 24(9):415-36, 2011.
76. Chaby, G., et al. “Refractory Venous Leg Ulcers: A Study of Risk Factors.” Dermatologic Surgery 32(4):512-9, 2006.
77. Neglén, P., Raju, S. “Intravascular Ultrasound Scan Evaluation of the Obstructed Vein.” Journal of Vascular Surgery 35:694-700, 2002.
78. Neglén, P., et al. “Stenting of the Venous Outflow in Chronic Venous Disease: Long-Term Stent-Related Outcome, Clinical, and Hemodynamic Result.” Journal of Vascular Surgery 46:979-90, 2007.
79. Kasirajan, K., et al. “Percutaneous AngioJet thrombectomy in the Management of Extensive Deep Venous Thrombosis.” Journal of Vascular and Interventional Radiology 12:179-85, 2001.
80. Kim, H.S., et al. “Adjunctive Percutaneous Mechanical Thrombectomy for Lower-extremity Deep Vein Thrombosis: Clinical and Economic Outcomes.” Journal of Vascular and Interventional Radiology 17: 1099-110, 2006.
81. Marston, W. Chapter 20—Venous Ulcers. In J.I. Almeida, ed. *Atlas of Endovascular Venous Surgery*, 2nd ed. Philadelphia, PA: Elsevier, 2019
82. Gloviczki, P., et al. “Reconstruction of Large Veins for Nonmalignant Venous Occlusive Disease.” Journal of Vascular Surgery 16:750-61, 1992.

83. Kistner, R.L. “Valve Reconstruction for Primary Valve Insufficiency.” In J.J. Bergan, R.L. Kistner, eds. Atlas of Venous Surgery. Philadelphia, PA: WB Saunders, 1992.
84. Raju, S. “Axillary Vein Transfer for Postphlebitic Syndrome.” In J.J. Bergan, R.L. Kistner, eds. Atlas of Venous Surgery. Philadelphia, PA: WB Saunders, 1992.
85. Kistner, R.L. “Transposition Techniques.” In J.J. Bergan, R.L. Kistner, eds. Atlas of Venous Surgery. Philadelphia, PA: WB Saunders, 1992.
86. Maleti, O., Lugli, M. “Neovalve Construction in Postthrombotic Syndrome.” Journal of Vascular Surgery 43:794-9, 2006.
87. Tripathi, R., et al. “Deep Venous Valve Reconstruction for Non-healing Leg Ulcers: Techniques and Results.” Australian and New Zealand Journal of Surgery 74:34-9, 2004.
88. Gloviczki, P., Bergan, J., eds. Atlas of Endoscopic Perforator Vein Surgery. London, UK: Springer-Verlag, 1998.
89. Tenbrook, J.A. Jr, et al. “Systematic Review of Outcomes after Surgical Management of Venous Disease Incorporating Subfascial Endoscopic Perforator Surgery.” Journal of Vascular Surgery 39:583-9, 2004.
90. Breu, F.X., Guggenbichler, S. “European Consensus Meeting on Foam Sclerotherapy.” Dermatologic Surgery 30(5):709-17, May 2003.
91. Frullini, A., Cavezzi, A. “Sclerosing Foam in the Treatment of Varicose Veins and Telangiectasia: History and Analysis of Safety and Complications.” Dermatologic Surgery 28:11-5, 2002.
92. Scurr, J.H. “Alternative Procedures in Deep Venous Insufficiency.” In J.J. Bergan, R.L. Kistner, eds. Atlas of Venous Surgery. Philadelphia, PA: WB Saunders, 1992.
93. Todd KL III, Wright, D.I.; VANISH-2 I Investigator Group. The VANISH-2 Study: A Randomized, Blinded, Multicenter Study to Evaluate the Efficacy and Safety of Polidocanol Endovenous Microfoam 0.5% and 1.0% Compared with Placebo for the Treatment of Saphenofemoral Junction Incompetence. Phlebology 29:608, 2014.
94. Almeida, J.I., et al. First Human Use of Cyanoacrylate Adhesive for Treatment of Saphenous Vein Incompetence. Journal of Vascular Surgery. Venous and Lymphatic Disorders 1:174, 2013.
95. Morrison, N., et al. Randomized Trial Comparing Cyanoacrylate Embolization and radiofrequency Ablation for Incompetent Great Saphenous Veins (VeClose). Journal of Vascular Surgery 61:985, 2015.
96. Toonder, I.M., et al. Cyanoacryulate Adhesive Perforator Embolization (CAPE) of Incompetent Perforating Veins of the Leg; A Feasibility Study. Phlebology 29:49, 2014.
98. Proebstle, T.M., et al. “Treatment of the Incompetent Great Saphenous Vein by Endovenous Radiofrequency Powered Segmental Thermal Ablation: First Clinical Experience.” Journal of Vascular Surgery 47:151-6, 2008.

97. Proebstle, T.M., et al. “Endovenous Treatment of the Greater Saphenous Vein with a 940 nm Diode Laser: Thrombotic Occlusion after Endoluminal Thermal Damage by Laser Generated Steam Bubbles.” Journal of Vascular Surgery 35:729-36, 2002.

16

Lymphedema: Complete Decongestive Therapy

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Jonathan A. Niezgoda, MA

Objectives

After completing this chapter, you will be able to:

- explain the anatomy and physiology of the lymphatic system
- describe the epidemiology of lymphedema
- differentiate between edema and lymphedema
- state the stages of lymphedema
- describe prevention and proper management of lymphedema.

Introduction

This chapter is possible because of the pioneers of lymphatic medicine who took special interest and development in the specialized treatment of lymphedema: Complete Decongestive Therapy (CDT).

The evolution of studying the lymphatic system and specialized treatment of the lymphatics began in Europe in the late 1800s (Fig. 16-1). The concept of utilizing a combined treatment protocol for lymphatic treatment, including “lymph massage,” compression and exercise emerged. In the 1930s, Dr. Emil Vodder,[1] developed and wrote on the specialized manual technique called “manual lymphatic drainage” (MLD). Professors Michael and Etelka Foldi are key German researchers and physicians that not only increased awareness of the lymphatic system and disorders associated with it but assisted in the foundation of the Gold Standard Treatment of Lymphedema, called Complete Decongestive Therapy (CDT).[2] They established the world renowned Foldi Clinic, a lymphedema treatment center, which now resides in Hinterzarten, Germany and authored the popular, *Foldi’s Textbook of Lymphology for Physicians and Lymphedema Therapists*.[2]

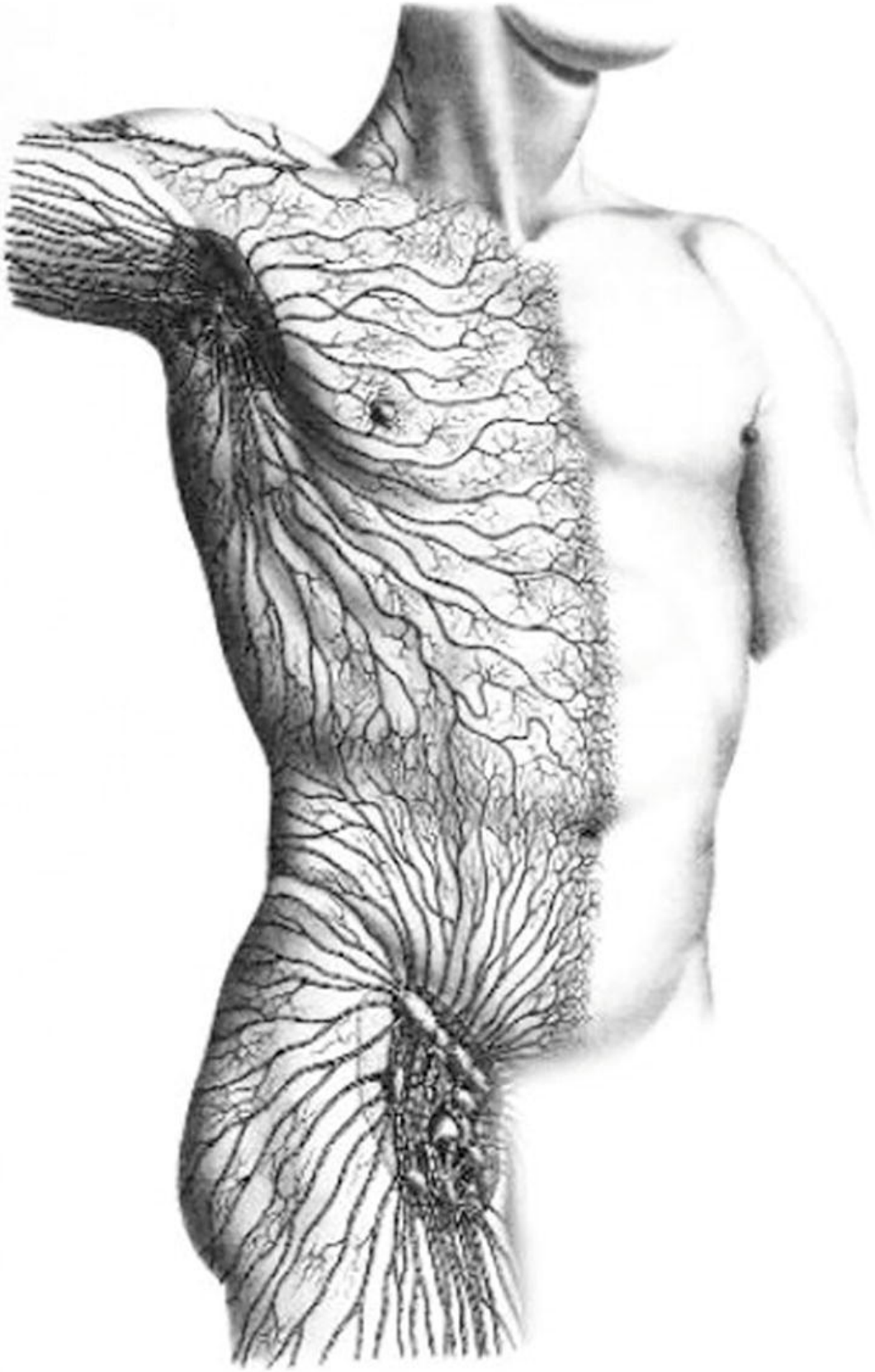


Figure 16-1. Anterior aspect of body. (18AD. https://chiklyinstitute.com/LDT. Accessed November 16, 2019.)

Guenter Klose is a pioneer of bringing CDT to the United States. Cultivating his skills at the Foldi clinic, Guenter assisted Dr. Lerner and later established a leading CDT school in the United States.[3] He is a leading expert, international instructor, and author on the lymphatic system and the treatment of chronic swelling disorders. Guenter along with the additional founders of their own CDT schools for lymphedema, Joachim Zuther, Steve Norton (international instructors and authors), and the Dr. Vodder School International formed NALEA (National American Lymphedema Education Association).[4] "These four schools provide instruction and training for the internationally recognized gold standard of CDT that is evidence based and medically sound."[4] The development of the Lymphology Association of North America (LANA)[5] in 1999 "promotes the standards for the certification of healthcare professionals who help individuals with lymphedema and/or related disorders manage their lymphedema and to promote lymphedema awareness and the science of lymphology."[5] It is also important to mention Saskia Thiadens, an RN, which in addition to all of her great efforts in the care and advocacy for those with lymphedema, founded the National Lymphedema Network (NLN).[6,7]

We gratefully acknowledge the contributions of Caroline Fife, MD for her work on previous editions of this chapter.

Definition of Lymphedema

“Lymphedema is a chronic inflammatory lymphostatic disease caused by the mechanical failure of the lymphatic system.”[2] This disease process may affect one or more parts of the body and disfiguring swelling can develop.

Role of the Lymphatic System

Acknowledging the lymphatic system is especially important as it serves immunological functions of creating and circulating white blood cells, detecting and combating foreign cells (Fig. 16-2). It helps establish homeostasis within the tissues by transporting fluids as well as removing molecular waste and proteins. Additionally, the lymphatic system transports and absorbs fats from the intestinal lymphatics.[3,8]

Figure 16-2. The role of lymphatics. (Welch, A.W. The Different Parts of the Immune System Play Unique Roles in Protecting the Body against Infection. 15AD. Available at: https://www.everydayhealth.com/news/10-amazing-facts-about-your-immune-system. Accessed December 26, 2019.)

Edema Versus Lymphedema

Edema and lymphedema may be used as generalized terms. Our goal in this chapter is to provide you with a foundation of understanding the lymphatic system, the distinct differences between edema and lymphedema, developing a skill set for assessing, and differentiating chronic swelling etiologies as well as understanding the international gold standard treatment approach for lymphedema.

Symptom Versus Mixed Disease Process

It is not uncommon for patients to present clinically with symptoms and implications related to edema formation. Although lymphedema is certainly not as common clinically seen as is edema, patients also present with pure forms of lymphatic dysfunction and disease with resultant lymphedema. Most commonly, patients who present with abnormal and excess tissue fluid are likely manifesting a mixed picture with components of both edema and lymphedema. Distinguishing edema from lymphedema can be challenging even for the experienced clinician. Thus, knowledge and understanding of both forms of dysfunction of tissue fluid formation is important as this serves as the basis for proper management.

Embryology

Embryology is the study of how the formation of the human body can impact different disorders. The embryology of the lymphatic system or lymphangiogenesis is still a controversial area with several theories that differ in the origin and pathways of the development of the lymphatic structure. There is consensus that the lymphatic system starts to form around the end of the 5th week, and most supported embryologic pathways suggests that the lymphatic system begins to form via paired jugular lymph sacs, followed by unpaired retroperitoneal lymph sac and then finally the paired posterior lymph sacs. With the exception of the anterior part of each lymph sac, each sac is invaded with mesenchymal cells, which then convert each lymph sac into lymph node groups. Then the spleen develops from these mesenchymal cells between the layers of the dorsal mesentery of the stomach (Fig. 16-3). It is clear that the lymphatic vessels arise from endothelial cell aggregates and that these lymph vessels appear later than blood vessel structures. The embryologic development of lymphangiogenesis is thought to be clinically relevant and an important pathway correlating with inflammation and wound healing.[3,8]

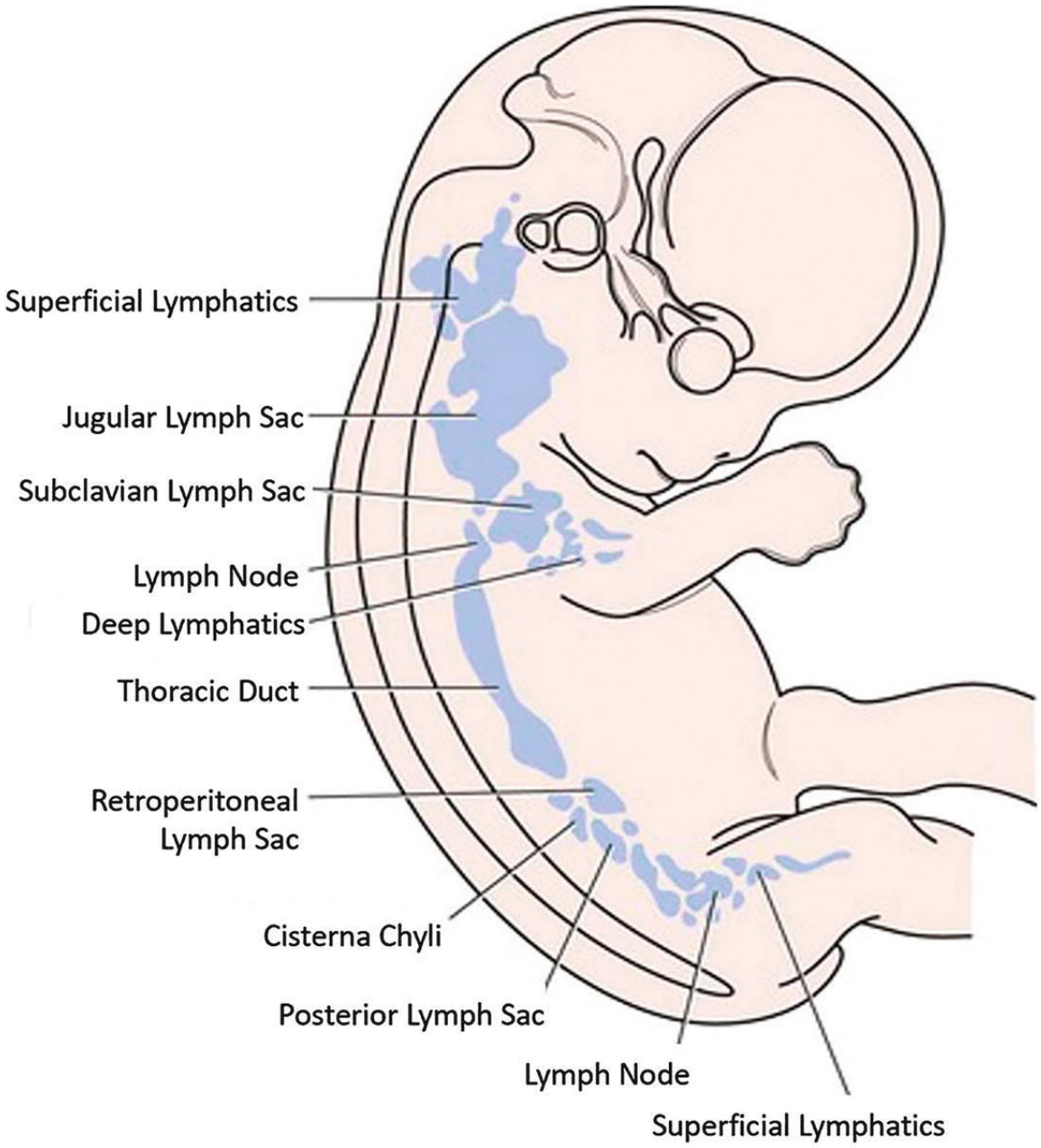


Figure 16-3. Embryology. (Welch, A.W. The Different Parts of the Immune System Plays Unique Roles in Protecting the Body Against Infection. Available at: www.everydayhealth.com/news/10-amazing-facts-abut-your-immune-system. Accessed December 28, 2019.)

Anatomy

The lymphatic system is a structured network of lymph vessels and organs that include lymph vessels (collectors), which carry lymph fluid from the interstitial tissue spaces to the central venous system. There are approximately 600 to 700 lymph nodes in the body; they create and store white blood cells and filter lymph fluid.[3,8] The spleen aids in filtering blood, recycles old red blood cells, and stores platelets and white blood cells. Part of the endocrine system, the thymus gland, develops and strengthens T lymphocytes (T cells) and serves an important role in immunity by defending the body against lethal pathogens. The thymus plays an important role early in life and begins to shrink after puberty. Tonsils also have immunological purpose, containing white blood cells, they are strategically located at the entrance of the body to aid in fighting bacteria entering the body. Lymphocytes, which are white blood cells and are in large quantities within the lymph nodes, travel throughout the blood, fighting off infections, bacteria, and foreign materials. Peyer's patches are located throughout the ileum within the small intestines. This unique lymph tissue assists in protecting against conceivably threatening foreign bodies that we are exposed to through the GI tract[3,8] (Fig. 16-4).

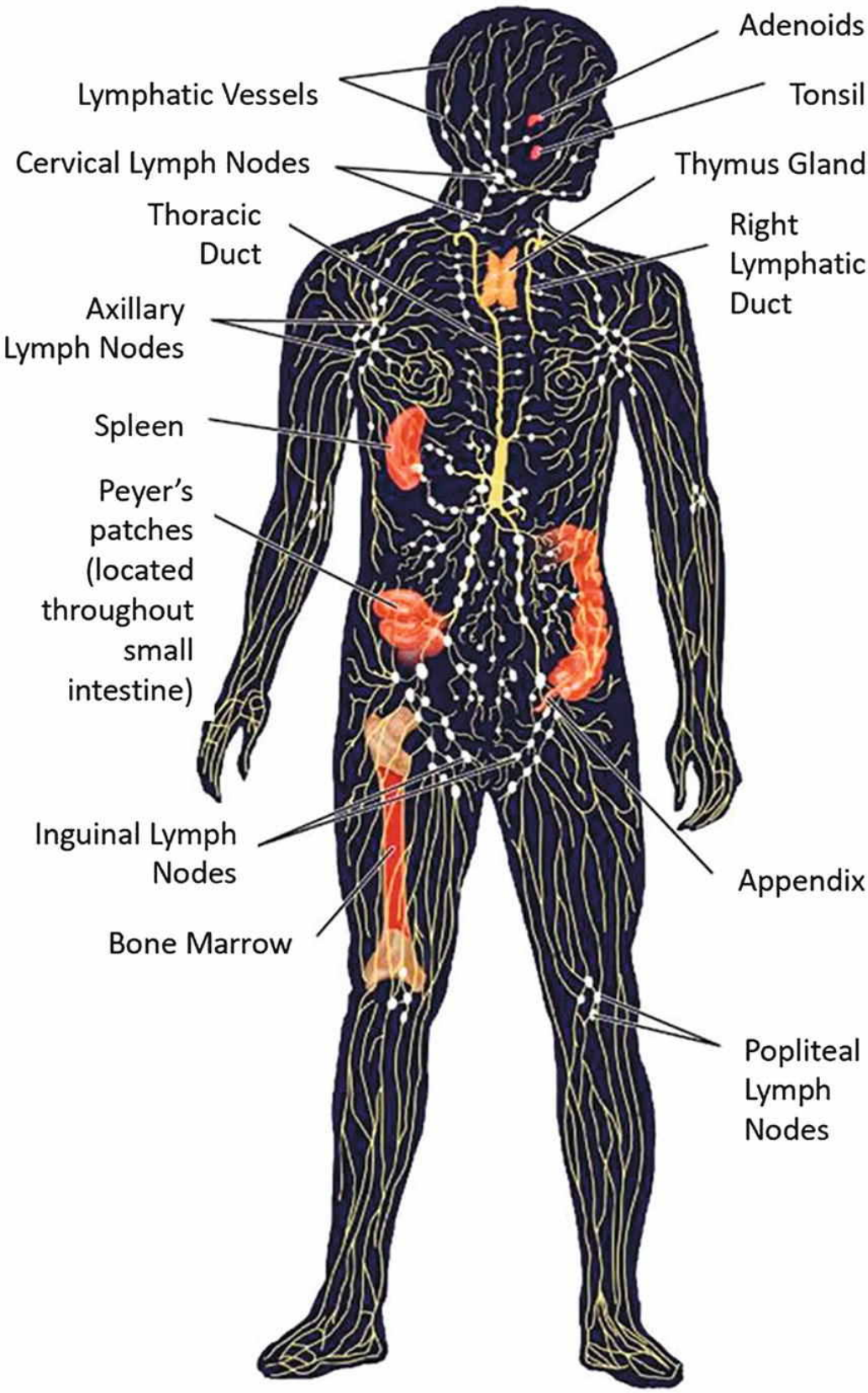

Adenoids
Lymphatic Vessels
Tonsil
Cervical Lymph Nodes
Thymus Gland
Thoracic Duct
Right Lymphatic Duct
Axillary Lymph Nodes
Spleen
Peyer's patches (located throughout small intestine)
Inguinal Lymph Nodes
Appendix
Bone Marrow
Popliteal Lymph Nodes

Figure 16-4. Anatomy. (The Human Lymphatic System, Showing the Lymphatic Vessels and Lymphoid Organs. *Encyclopædia Britannica*. 2000. Available at: http://www.citationmachine.net/american-medical-writers-association-journal/cite-a-image/manual. Accessed November 16, 2019.)

Fascia separates the deep and superficial layers of the lymphatic system. The suprafascial layer drains the lymphatic fluid from the skin into the subcutaneous tissue. The subfascial layer of the deep lymphatic system then drains the lymph from muscle and nervous tissue, tendon sheaths, periosteum, and joints. However, there are some distally located joints on the extremities that drain from the superficial layer. Encapsulated within the subcutaneous fatty tissue, transport vessels are stationed within the superficial lymphatic system and the transport vessels of the deep system, which follow the blood vessels, are within the deep membrane. Connecting the deep and superficial systems are perforating vessels. The lymph organs described previously are part of the deep system[8–12] (Figs. 16-5 to 16-7).

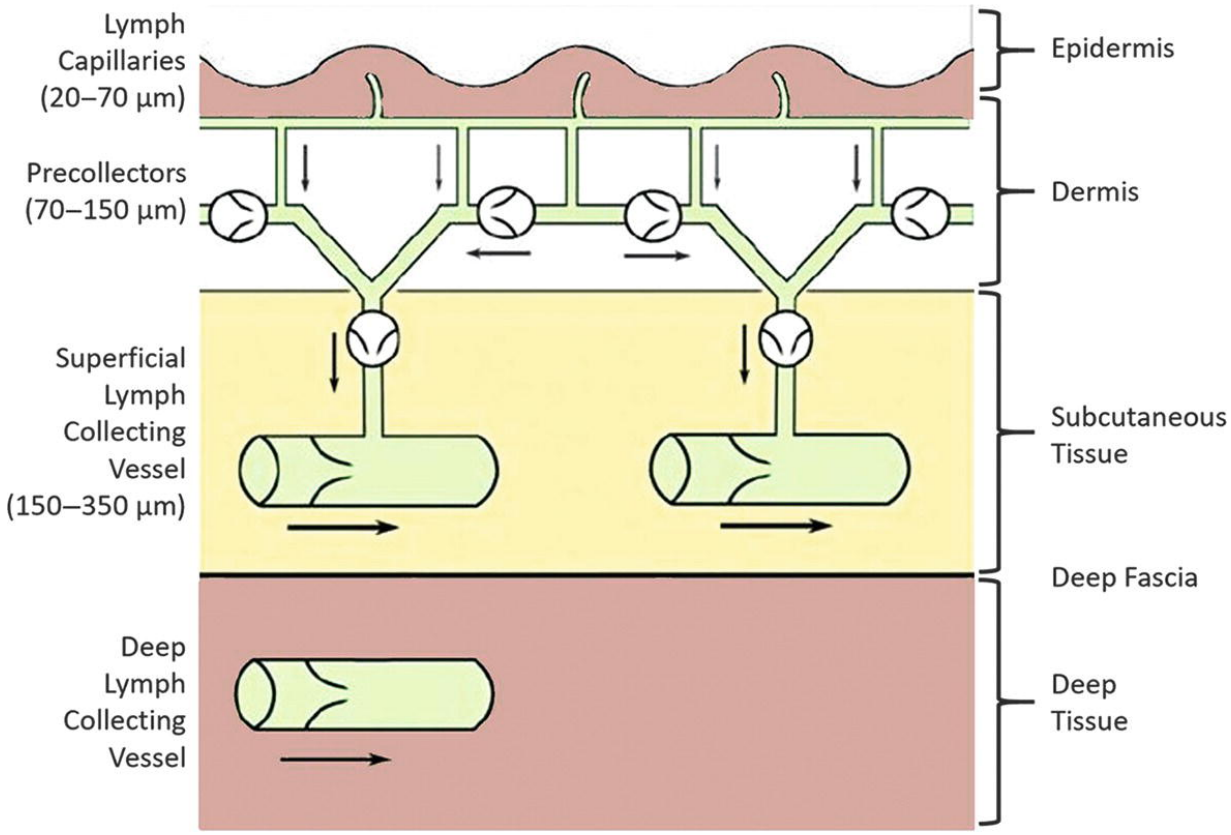


Figure 16-5. Lymphatic transport vessels. (Suami, H. *FIG. 2. Schematic Diagram of the Relationship between the Lymph Capillaries, Precollectors, and Lymph Collecting Vessels.* Research Gate. Available at: https://www.researchgate.net/publication/5803842_The_Lymphatic_Anatomy_of_the_Breast_and_its_Implications_for_Sentinel_Lymph_Node_Biopsy_A_Human_Cadaver_Study. Accessed November 16, 2019.)

Figure 16-6. Lymphatic capillaries in feet. (*Ref. Picture: 08504*. Available at: https://www.biusante.parisdescartes.fr/ressources/php/fragments/banque_images_ajax_proxy.php?do=informations-iconographiques&refphot=08504. Accessed November 16, 2019.)

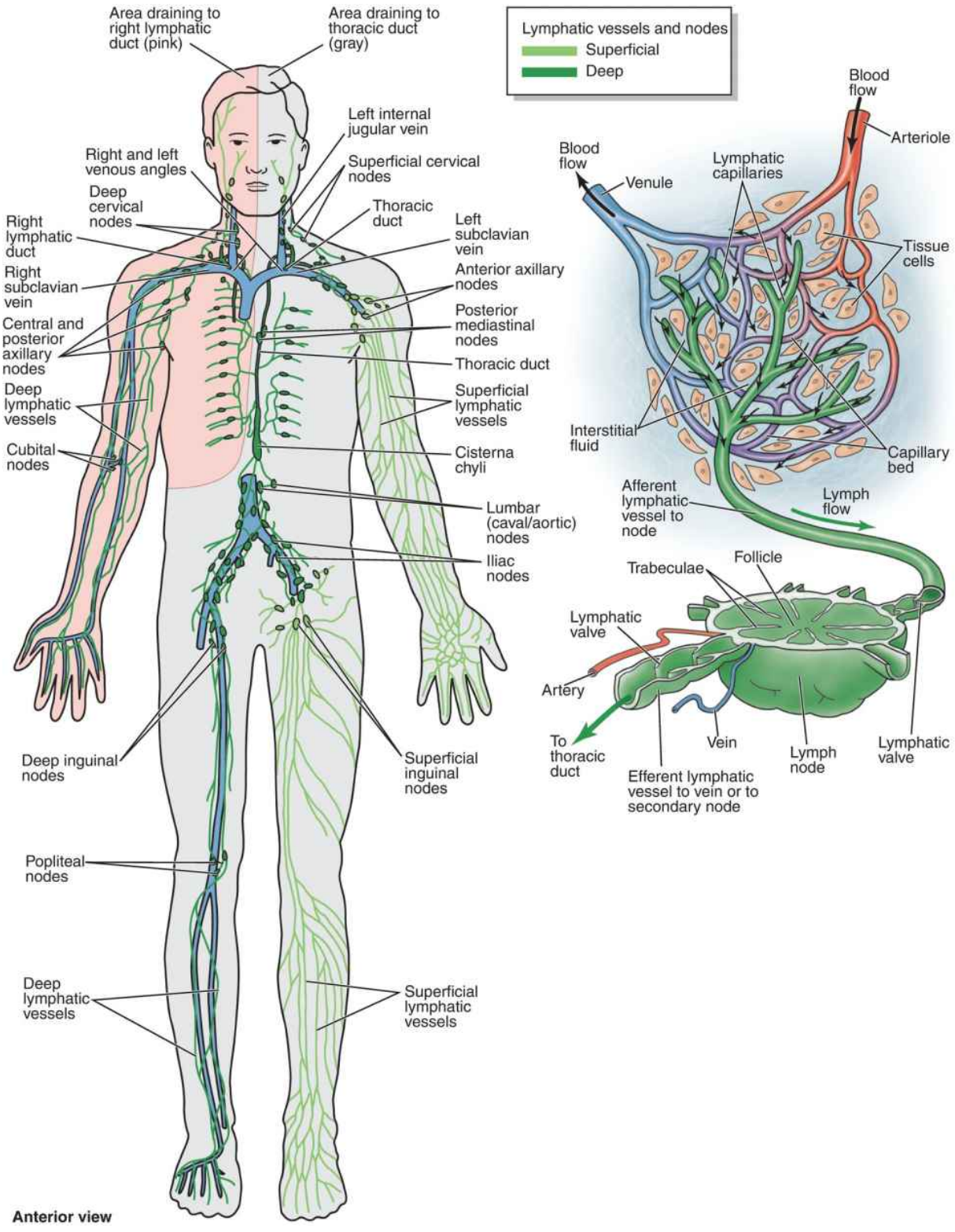


Figure 16-7. Lymphatic vessels (superficial and deep system). (Used with permission from Wound, Ostomy

and Continence Nurses Society®; Doughty, D.B., McNichol, L.L. *Wound, Ostomy and Continence Nurses Society® Core Curriculum: Wound Management*. 2015.)

Lymph Fluid and Fluid Capillaries

Lymph fluid is a clear fluid made up of water, red blood cells, white blood cells (exclusively lymphocytes), water, proteins, fatty acids and waste products (contain foreign substances), cell particles, and cancer cells. Intestinal lymph, known as chyle, is thought of as “milky white” in color and is made up of long-chain triglycerides and vitamins A, D, E, and K. They are absorbed by the lymphatics and are transported from the intestine to the liver. Foldi deemed the name “lymphatic load,” which is the explanation for the substances leaving the interstitial tissue spaces via the lymphatic system.[2] Daily, 75 to 100 g of proteins are carried by the lymph vessels, which is about half the amount of proteins circulating throughout the body.[3,12]

The tree-like system of the lymphatics begins as small branches within the superficial system and grow to large trunks within the deep system. In sequential order, they are known as the lymph capillaries, precollectors, collectors, and trunks[13] (Fig. 16-7).

Lymph capillaries are a complex network of vessels beginning in the dermal layer that are strategically close to the blood capillaries, covering the entire body. They are more permeable and are larger than blood capillaries, allowing for the absorption of macromolecules such as proteins and other fluids to be reabsorbed into the venous system. The lymph capillaries are made up of a flat layer of endothelial cells and are connected to the interstitial tissue by “anchoring filaments,” which allow the vessels to stay open during varying pressure gradients within the interstitial tissue space. Since lymph capillaries do not have valves, the “swinging flaps” of the anchoring filaments open the lumen of the lymph capillaries so that the interstitial fluid may be drained.[13] The initial vessels account for approximately a 3 to 4 cm area in circumference. More condensed in the plantar areas of the feet and palmar areas of hands and digits, the circumference decreases to a circumference of 1.5 to 2 cm. Therefore, when increased swelling occurs, it is more prevalent in the dorsal aspects of hands and feet[8] (Fig. 16-8).

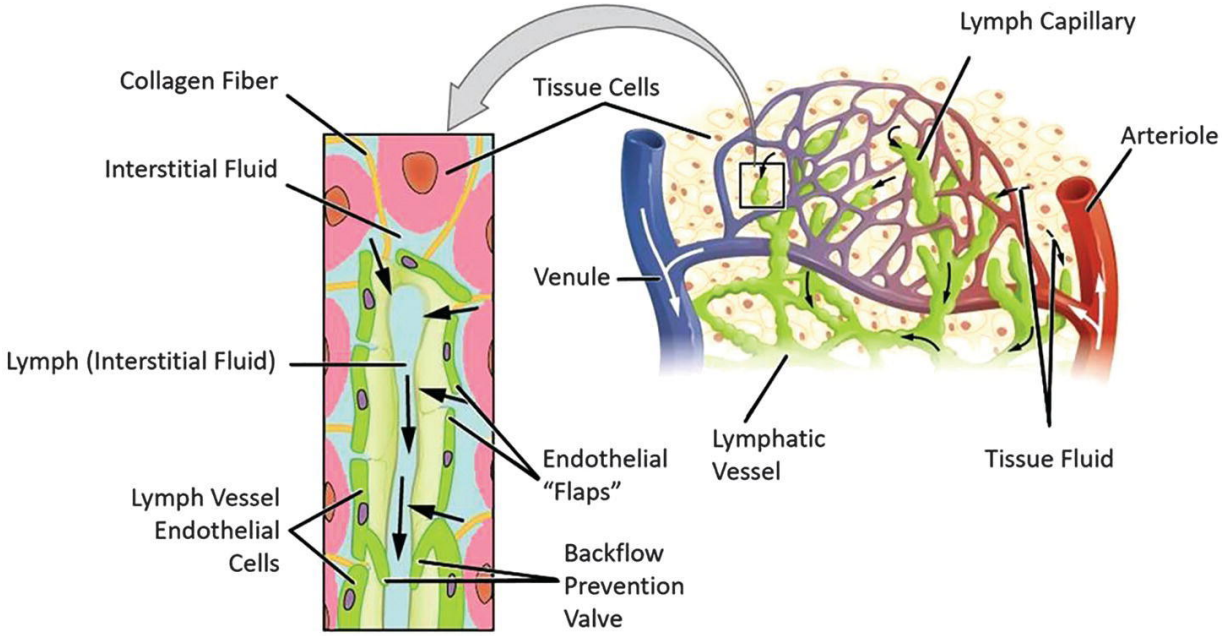


Figure 16-8. Lymph capillaries in the tissue spaces. (*Wikkimedia Commons*. 2017. Available at: https://commons.wikimedia.org/wiki/File:2202_Lymphatic_Capillaries.jpg. Accessed November 16, 2019.)

Lymph Collectors

Precollectors connect the lymph capillaries to the collector vessels. Some are made of smooth muscle and contain valves, which regulate directional flow, channeling lymph fluid into the collectors. Others have absorptive qualities like the capillaries. Perforating precollectors create a connection between the superficial and deep lymphatic system by penetrating through the fascia. For example, they are an important aspect when considering MLD of an extremity as they direct fluid from superficial aspects of the extremity to the deep truncal aspect. The perforating precollectors are passive and do not demonstrate activity until they are under high intralymphatic pressure, when they become the direct network between the superficial and deep lymphatic system.[8,10–13]

Lymph collectors are responsible for transporting lymph fluid from body areas and passing it along to the lymph nodes and then on to the lymphatic trunks. In structure, they resemble veins but have thinner walls and the valves are closer in proximity to prevent fluid backflow and provide directional transport of lymph fluid from distal to proximal as well as to the regional lymph nodes. The collector vessels range in size from 0.1 to 0.6 mm, and depending on the diameter of each vessel, the bicuspid valves are every 0.6 to 2 cm in the collectors and 6 to 10 cm in the thoracic duct.[3]

Positioned between the proximal and distal pair of valves are lymphangions, which are made up of smooth muscle tissue, receptors, and bicuspid valves. The collectors are known to resemble a “pearl necklace” due to the structural appearance of the lymphangions. The “caterpillar type” movement that takes place during lymphangiomotoricity, under normal conditions, occurs 10 to 12 times per minute.[12] Contraction of the lymphangions are prompted by diaphragmatic breathing, muscle activity, pumping of the arteries, negative pressure in the central veins, and external compression (e.g., MLD).[3] Lymphangiomotoricity increases when lymph volume increases and lymphangions are able to contract up to 60 times per minute.[3,8] “Other factors that increase contraction frequency include stretch on the wall of the lymphangion (effect of MLD), temperature,

activity of muscles and joints, diaphragmatic breathing, pulsation of adjacent arteries, and certain tissue hormones”[13] (Fig. 16-9).

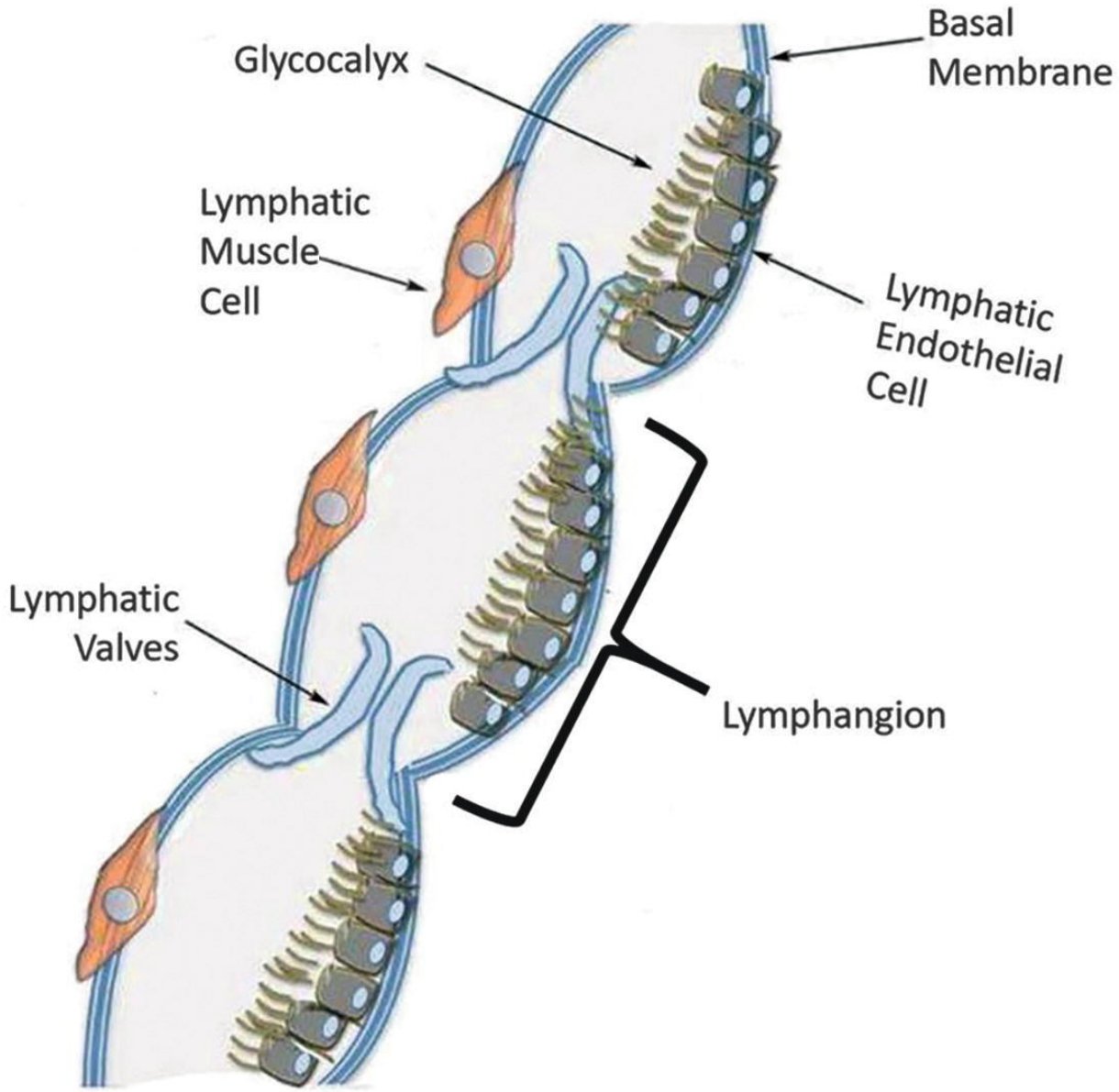


Figure 16-9. Lymphangion. (Hansen K, D’Alessandro A, Clement C, et al. “Lymph Formation, Composition and Circulation: A Proteomics Perspective.” *International Immunology* 27(5):219-27, May 2015; Modified and enhanced art by Jonathan Niezgoda.)

Lymphatic Trunks and Ducts

Lymphatic trunks and ducts are the largest vessels in the lymphatic system and collect fluid from the organs, extremities, and related quadrants of the trunk. They are innervated predominantly by the sympathetic component of the autonomic nervous system and have the same muscle wall and valve composition as the collectors; however, the structures are stronger.[8] The lymphatic trunks merge and form ducts, which allow the transport of lymph fluid into the venous circulation.

Approximately 0.2 to 0.5 mm in diameter and 40 to 45 cm in length, the thoracic duct is the largest lymphatic vessel in the human body. It runs parallel to the spine from L2 to the venous angle (the juncture between the left internal jugular and the left subclavian veins) emptying approximately 3 L of lymph fluid into the venous angle daily. It is responsible for draining ¾ of the body's lymph. It perforates the diaphragm and runs to the base of the neck. Lymph fluid velocity and flow increase with diaphragmatic (abdominal) breathing, an important component in the stimulation of lymph fluid. Due to its great length, the thoracic duct is subdivided into the abdominal, thoracic, and cervical parts. The cisterna chyli is a sack-like formation at the abdominal portion of the thoracic duct.[3,8,13]

The lymphatic trunks of the lower body are the right and left lumbar trunks and the intestinal trunk. The lymphatic trunks of the upper body include right and left jugular trunks, right and left subclavian trunks, and right and left bronchomediastinal trunk. The remaining ¼ of lymph is drained via the right lymphatic duct into the right venous angle/subclavian vein[3,8] (Fig. 16-10).

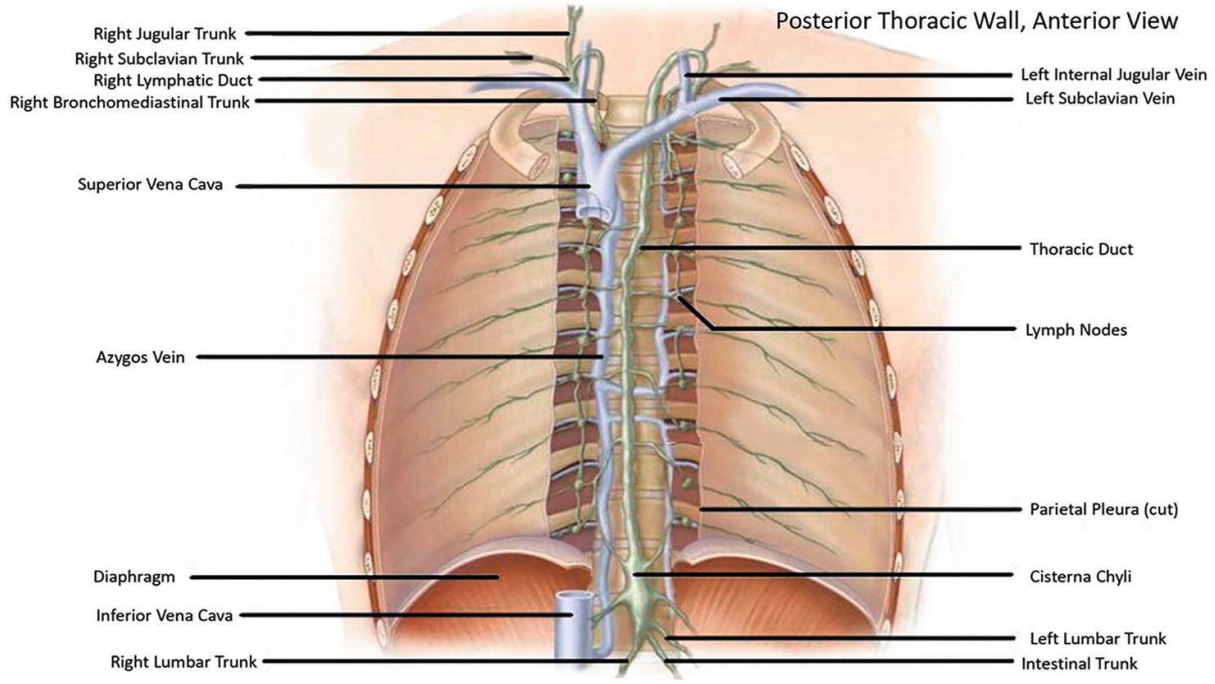


Figure 16-10. Lymphatic trunks. (https://antranik.org/the-lymphatic-system/. Accessed November 20, 2019.)

Lymph Nodes

Lymph nodes drain and filter the waste from lymph fluid, which is delivered by the lymphatic collectors. There are approximately 600 to 700 lymph nodes in the human body. A vast majority of lymph nodes dwell within the head/neck and intestines as these areas are passageways for pathogens. They vary in shape and can be 0.2 to 3 cm in length. Lymph nodes can harden with age, which can cause swelling. Regional lymph nodes play a role in the body's first line of defense[8] (Fig. 16-11).

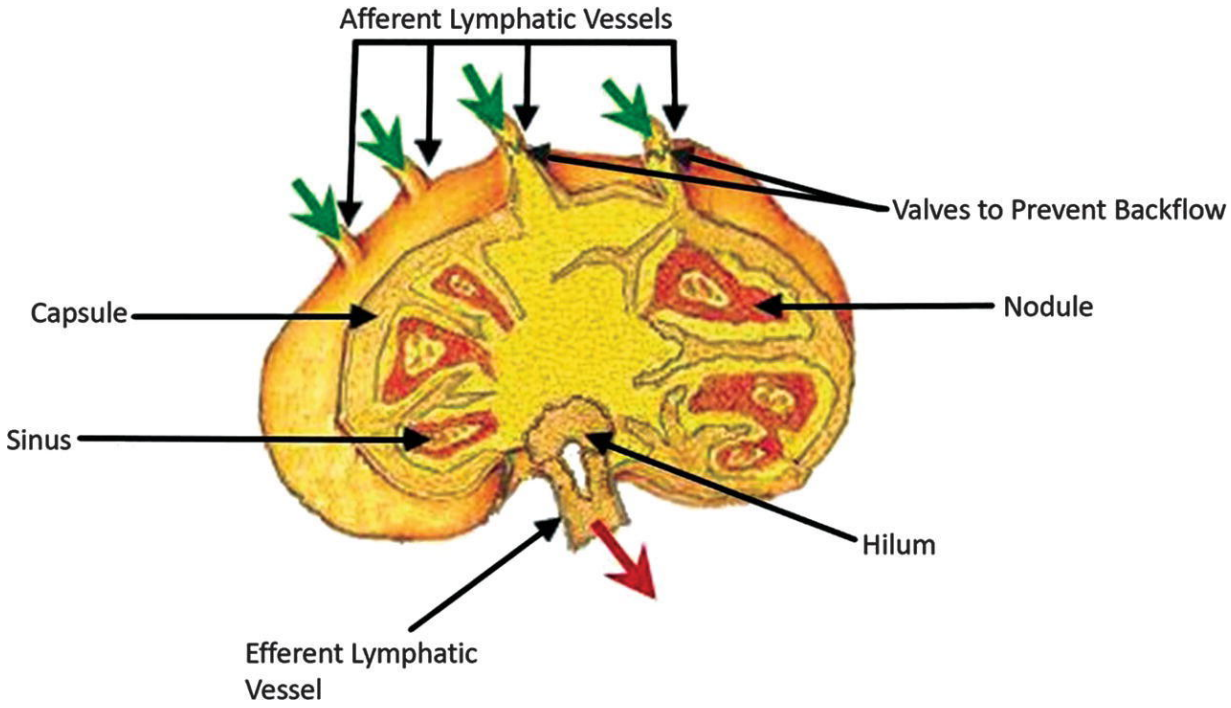


Figure 16-11. Lymph node structure. (*National Cancer Institute SEER Training Modules*. Available at: https://training.seer.cancer.gov/anatomy/lymphatic/components/nodes.html)

Lymph nodes have three main purposes including[9]:

1. Filtration of pathogens and destructive matter including bacteria, cancer cells, toxins, and dead cells.
2. Immunological component produces and stores lymphocytes. Within the lymph nodes, antibodies are produced, carried out by the efferent lymph collectors, and are distributed in the bloodstream.
3. The blood capillaries within the lymph nodes reabsorb water, therefore regulating the concentration of proteins.[3,12,13]

Lymphatic Watersheds and Lymphatic/Interterritorial Anastomoses

"Lymphatic watersheds represent linear areas on the skin that separate territories from each other and contain relatively few lymph collectors."[12] They are instrumental in labeling lymphatic drainage routes (Fig. 16-12).

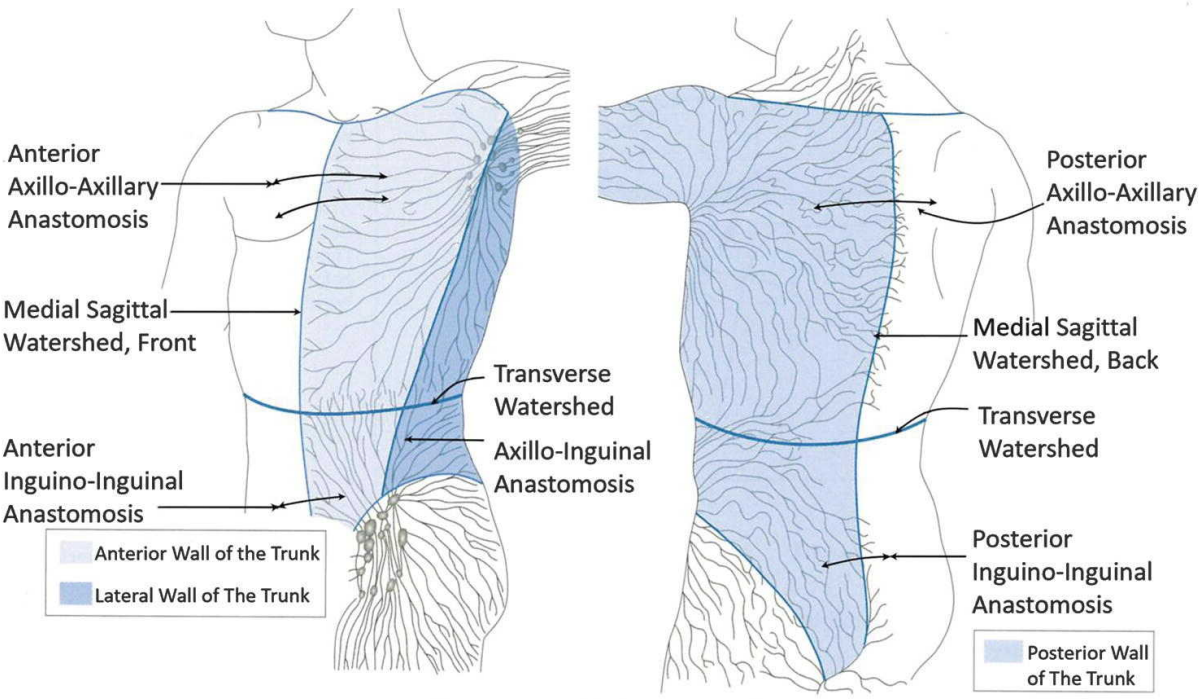


Figure 16-12. Lymphatic watersheds. (Used with permission. Zuther/Norton textbook.)

Lymphatic/interterritorial anastomoses are the connections between lymph collectors going across a watershed. They are used in manual lymphatic drainage to move fluid from a swollen, congested area to an unaffected part of the body. Typically inactive, they are a pathway of approximately 7 to 10 collectors in each area. By understanding the locations of anastomoses, lymph fluid may be redirected from an area of dysfunction toward an unaffected area.[3,12]

Physiology

The lymphatic system should be recognized as an individual system within the human body. Its purpose includes the following:

- Fluid homeostasis: Regulating pressures within the interstitial tissue spaces by collecting and transporting fluid back to the venous system (e.g., plasma proteins).
- Immunological: Produces antibodies and protects the body from infection.
- Transport of nutrients: Absorbs and transports essential nutrients from the intestinal lymphatics.

Lymphatics function is based on fluctuating pressures within the interstitial tissue space. In addition to its immunological role and assisting with surveillance, it shares a close working relationship with the cardiovascular system in transporting and filtering interstitial fluid.[3,8,12]

The cardiovascular or circulatory system is a closed pathway. The lymphatic system, known as a “one-way” system with an open pathway, transports lymph fluid from the interstitial tissue spaces back into the vascular system. Unlike the circulatory system that has a central pump (heart), the lymphatic system is a complex network of pathways with the lymphatic capillaries housing lymphangions, small pumping “hearts”[3,12] (Fig. 16-13).

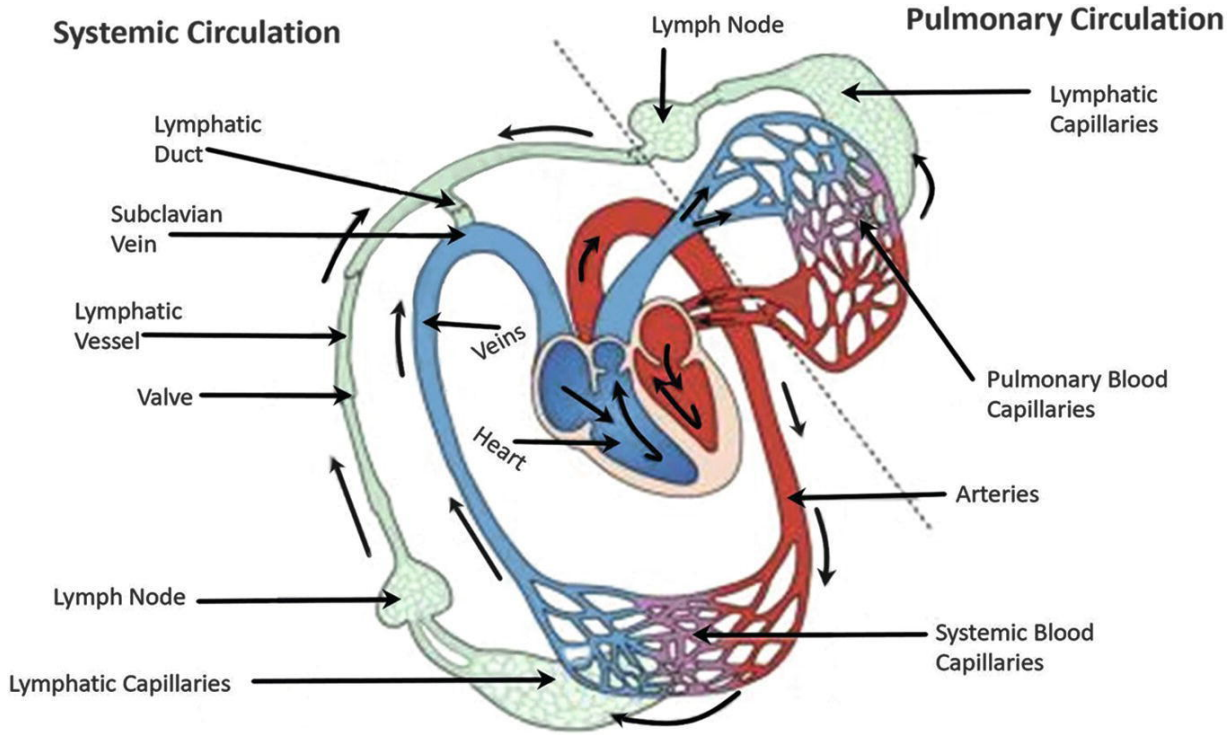


Figure 16-13. Lymphatic and pulmonary circulation. (*Chapter 22 The Lymphatic System*. Available at: https://pdfs.semanticscholar.org/7bf7/f080670b944477777ea8e39383187e244130.pdf)

In considering fluid transport and exchange, it is important to understand that the human body is made up of the intravascular and extravascular fluid volume compartments. The continual transfer of fluids, gases, and nutrients that occurs between these two compartments is at the blood capillary level. The physiological components responsible for capillary exchange are diffusion, osmosis, filtration, and reabsorption[8,12] (Fig. 16-14).

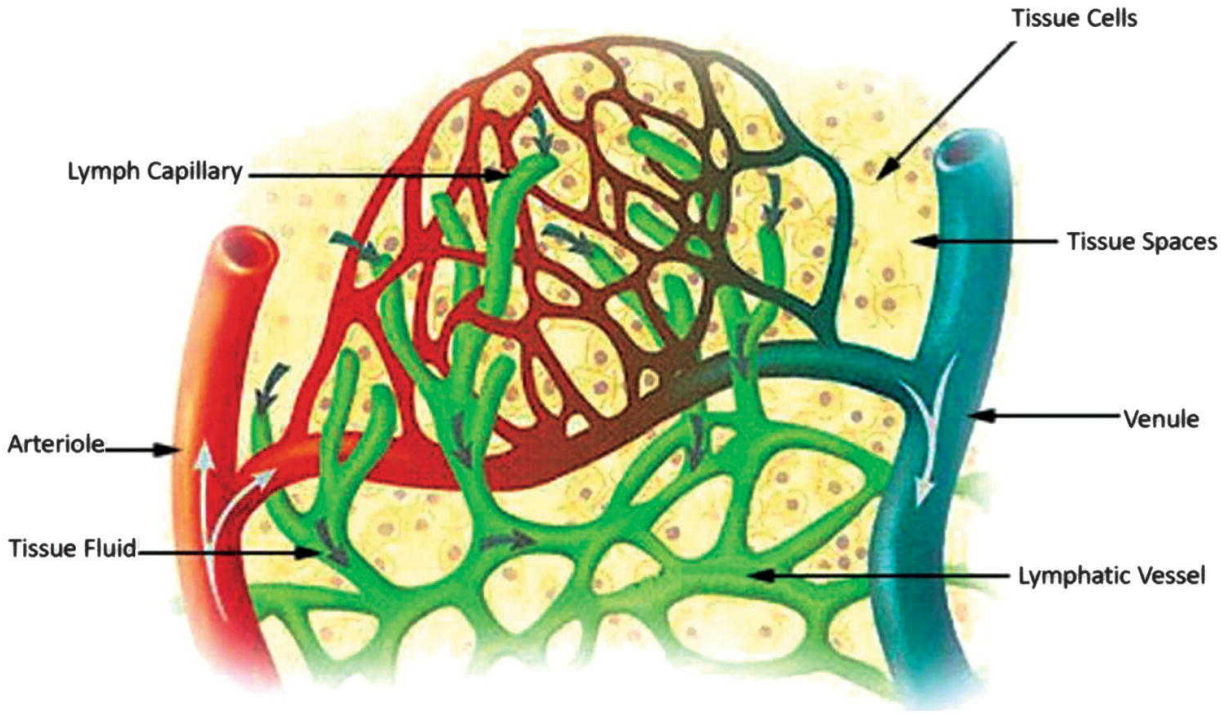


Figure 16-14. Lymphatic capillaries in tissue spaces. (*National Cancer Institute SEER Training Modules*. Available at: https://training.seer.cancer.gov/anatomy/lymphatic/components/)

The interstitium makes up nearly 1/6 of the human body. It consists of proteoglycan filaments, collagen fiber bundles, and fluid, which are thought of as the “glue” that holds cells together. The interstitial space is able to withstand up to 30% more water before swelling is observed. Thus, a patient may subjectively complain of a limb or part of the body “feeling heavy,” even though swelling is not clinically evident.[3]

Ernest Starling, a British physiologist, developed the Starling’s Equilibrium for Capillary Exchange in 1896.[14] He hypothesized that under ordinary circumstances, fluid exiting the arterial side of capillaries “almost” equals the fluid that is reabsorbed back into circulation. Starling noted the lymphatic system being responsible for the amount of fluid that was left from the disequilibrium or imbalance. Initially, it was thought the amount of fluid uptake by the lymphatics

was approximately 2 to 4 L/d.[3] The new Revised Starling's Law suggests an even greater amount of fluid uptake by the lymphatics.[14]

The glycocalyx (Fig. 16-15) is a layer outside the cell, which plays a role in fluid exchange in between the cell and the interstitial fluid and it also acts as a protective layer defining what types of proteins can interact with a given cell. The presence and function of the glycocalyx has played a very significant role in the revision of Starling's equation, changing the absorption pressures from interstitial oncotic pressure to subglycocalyx oncotic pressure.[14] This changed the popular assumption that sustained venous absorption accounted primarily for tissue fluid balance to the theory that subglycocalyx oncotic pressure increases so rapidly that it prevents sustained absorption into the venous system, meaning that there is a net increased load on the lymphatic system to move fluid. Also, the assumption about interstitial oncotic pressure was dramatically overestimated since it could not be directly observed, which in turn influenced the net filtration rate force and the amount of lymph production.[14]

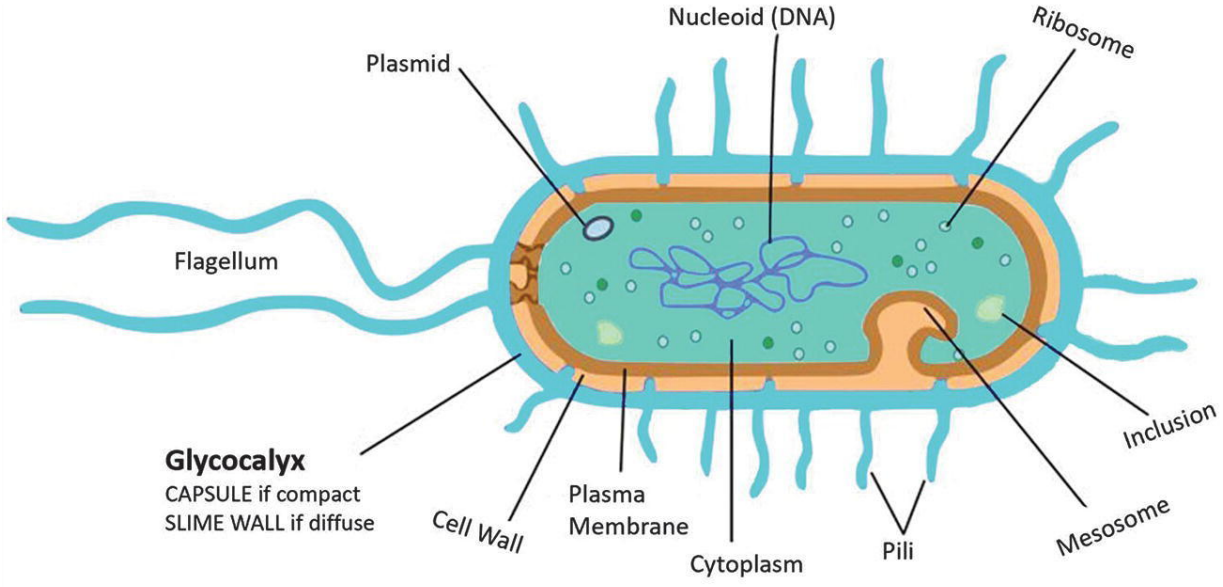


Figure 16-15. The glycocalyx. (P. *WPClipArt*. Available at:

https://www.wpclipart.com/medical/microscopic/bacteria/bacteria_cell_diagram.png.html)

Lymphatic Impairment

Edema is a symptom resulting from a variety of etiologies, which will be further discussed later in the chapter. Lymphedema is a disease process that is the result of lymphatic impairment. Edema or lymphedema can result from dynamic, mechanical, and combined insufficiency.

Dynamic insufficiency or high-volume insufficiency occurs when a healthy lymphatic system is overwhelmed. The lymphatic load exceeds the transport capacity. Because of the increased filtration from the blood capillaries, the edema is indicated by increased interstitial fluid due to high fluid and low protein. Examples of dynamic insufficiency include edema as a result of congestive heart failure (CHF), chronic venous insufficiency (CVI), postthrombotic syndrome, and dependency. Hypoproteinemia allows increased filtration out of the capillaries due to low plasma proteins; examples include kidney disease, diseases of the liver, starvation, and anemia.[12,13]

If edema resulting from dynamic insufficiency persists untreated, impairment to the lymphatic vessel walls and valves occurs as a result of unanswered increased transport requirements and resultant intralymphatic pressure in the lymph capillaries. To avoid secondary lymphedema, modalities to treat the swelling include elevation, ambulation, and compression. MLD is not contraindicated as it will not increase the transport capacity. In cases of edema resulting from CHF, diuretics and treatment of underlying heart condition are also indicated.[12,13]

Mechanical insufficiency or low-volume insufficiency occurs due to a mechanical failure within the lymphatics. One reason for the decrease in transport capacity is due to secondary causes of damage such as surgery, radiation, trauma, and infection. Congenital malformation or absence of lymphatic tissue is another cause of mechanical insufficiency, called primary lymphedema.[12,13] Lymphatic treatment is warranted in cases of lymphatic mechanical dysfunction.

Combined insufficiency occurs when there is both a decrease in transport capacity and an increase in lymphatic load: dynamic and

mechanical insufficiency are occurring at the same time.[13] Phlebolymphedema is an example of combined insufficiency.

Phlebolymphedema, caused by CVI, results in localized hemodynamic pathology. If CVI is not appropriately diagnosed and treated, high volumes of fluid leak into the interstitial tissue space, causing increased edema and lymphatic insufficiency. The dynamic insufficiency can be further complicated by mechanical insufficiency of the lymphatics as the increased levels of tissue proteins cause chronic fibrosis of the lymphangion walls as well as damaging the lymphatic vessels. In advanced stages of CVI, significant fibrotic changes occur, destroying the lymphatic channels and obstruct lymph flow. Complete decongestive treatment is beneficial in cases of advanced phlebolymphedema.[8,12,13]

Lipedema is the deposition of excess fat in subcutaneous tissues. Lipedema usually does not affect the feet, whereas lymphedema does, and lipedema usually presents symmetrically across both legs.

Active hyperemia/passive hyperemia/hypoproteinemia: In active hyperemia, there is increased organ blood flow that is associated with increased metabolic activity and increases in lymphatic load through an increase in capillary pressure. Passive hyperemia occurs when blood flow cannot properly exit an organ and then builds up in the surrounding blood vessel, which increases blood capillary pressure which increases lymphatic load. Hypoproteinemia is a condition where there is an abnormally low level of protein in blood, several causes all result in edema once serum levels fall below a certain level. This reduces absorption into venous capillaries, which in turn increases lymphatic load.[3,8]

Lymphatic Classifications

Lymphedema is classified as primary or secondary and can develop anywhere in the body. It is often noted in extremities, but lymphatic impairment can also occur in the head/face/neck, trunk, genitalia, intestines, and chest/breast areas.[3,8]

Primary Lymphedema

In primary lymphedema, there are three types of malformation and dysfunction of the lymphatics. They include hypoplasia (decreased amount of lymph vessels), hyperplasia (increased amount of lymph vessels with impaired function), and aplasia (absence of lymph vessels). Common syndromes of primary lymphedema include Klippel-Trenaunay Syndrome, Noonan's Syndrome, Parkes Weber Syndrome, Prader-Willi Syndrome, and Turner's Syndrome.[3] The National Organization for Rare Disorders (NORD)[15] provides information on all syndromes related to primary lymphedema.

Secondary Lymphedema

Secondary lymphedema occurs due to an insult or damage to the lymphatic system. In the United States, lymphedema is often associated with breast cancer, mastectomy, and surgical excision of lymph nodes.[16] Additional causes of lymphedema include CVI, cancer treatment, burns, orthopedic trauma or procedures, obesity, vascular bypass, infection, wounds, and vein harvesting for coronary artery bypass procedures.

Filariasis is the most common cause of secondary lymphedema in eastern developing countries. An infection caused by parasites, filariasis damages the lymph vessels and nodes.[13] Filariasis is considered to be a neglected tropical disease and the most frequent cause of permanent disability worldwide.[13]

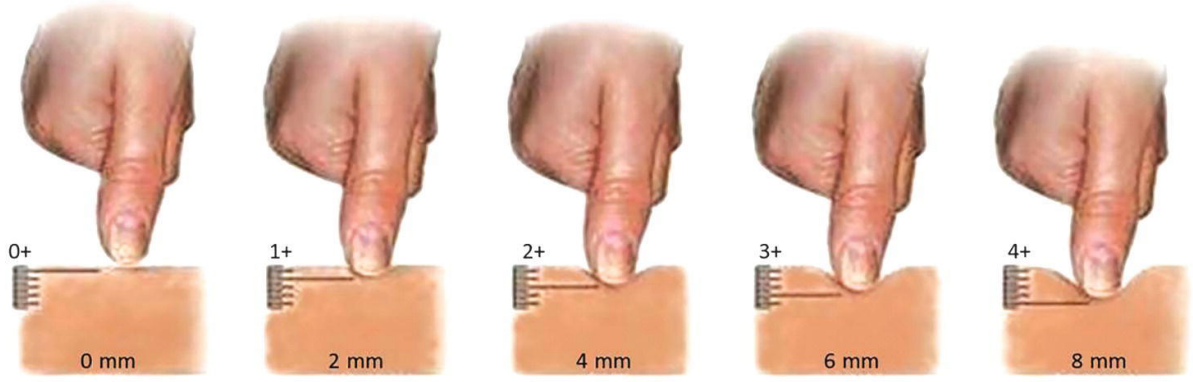
Characteristics of Lymphedema

Discomfort

Patients often provide subjective complaints when discussing swelling associated with their lymphedema: “tightness, heaviness, achiness, increased feeling of tension to the affected area.” Severe pain isn’t typically associated with lymphedema diagnosis; however, it can be associated with infection/cellulitis, malignancy, neurological, and/or orthopedic issues.[3,12]

Pitting Edema

Pitting edema is present in early stages of lymphedema. As the stages of lymphedema progress, fibrosis and other skin changes and breakdown occur (Fig. 16-16).



0+ No Pitting Edema
1+ Mild Pitting Edema, 2 mm depression that disappears rapidly
2+ Moderate Pitting Edema, 4 mm depression that disappears in 10–15 seconds
3+ Moderately Severe Pitting Edema, 6 mm depression that may last more than 1 minute
4+ Severe Pitting Edema, 8 mm depression that can last more than 2 minutes

Figure 16-16. Pitting edema. (*Knowledge Update*. 2015. Available at: http://siddiquiphysio.blogspot.com/2015/03/grades-of-edema-easy-way-to-remember.html)

Infection

Due to the intimate relationship between the immune system and the lymphatic system, a diminished immune response coupled with the high protein content of the interstitial fluid creates ideal conditions for bacterial growth. These infections can include local wound infection or cellulitis, as well as deeper and systemic infections. Such infections may be recurrent and can occur without skin ulcers being present.

Stemmer's Sign

A positive Stemmer Sign (performed by pinching the skin at base of digit or affected area such as the base of the second toe or middle finger) indicates the presence of lymphedema. If when pinched the skin does not lift up, the sign is positive. If you can pinch and lift the skin, the Stemmer's sign is negative (Fig. 16-17).

Figure 16-17. Stemmer's sign. (AZH Wound and Vascular Center, Milwaukee, Wisconsin; Photo courtesy of J. Niezgoda.)

Presentation/Skin Changes

Patients with lymphatic insufficiency may present with a variety of dermal findings. Fibrotic skin changes, such as fissures, together with hyperkeratosis, papillomatosis, and increased surface debris and scale, elevate the possibility of bacterial or fungal colonization or invasion. Furthermore, in patients with wound or chronic ulcers, increased edema is associated with higher rates of wound drainage, which means clinicians need to be cognizant about the risk of skin maceration, more frequent dressing changes, selection of more absorptive dressings, and appropriate bandaging. Examples of skin and dermal manifestations of lymphedema include:

- Hyperkeratosis (Fig. 16-18)

Figure 16-18. Hyperkeratosis. (AZH Wound and Vascular Center, Milwaukee, Wisconsin; Photo courtesy of J. Niezgoda.)

- Papillomatoses (Fig. 16-19)

Figure 16-19. Papillomatoses. (AZH Wound and Vascular Center, Milwaukee, Wisconsin; Photo courtesy of J. Niezgoda.)

- Erythema (Fig. 16-20)

Figure 16-20. Erythema. (AZH Wound and Vascular Center, Milwaukee, Wisconsin; Photo courtesy of J. Niezgoda.)

- Open ulcers (Fig. 16-21)

Figure 16-21. Open ulcers. (AZH Wound and Vascular Center, Milwaukee, Wisconsin; Photo courtesy of J. Niezgoda.)

- Obesity (Fig. 16-22)

Figure 16-22. Obesity. (AZH Wound and Vascular Center, Milwaukee, Wisconsin; Photo courtesy of J. Niezgoda.)

- Fibrosis (Fig. 16-23)

Figure 16-23. Fibrosis. (AZH Wound and Vascular Center, Milwaukee, Wisconsin; Photo courtesy of J. Niezgoda.)

- Sclerosis/fibrosis (Fig. 16-24)

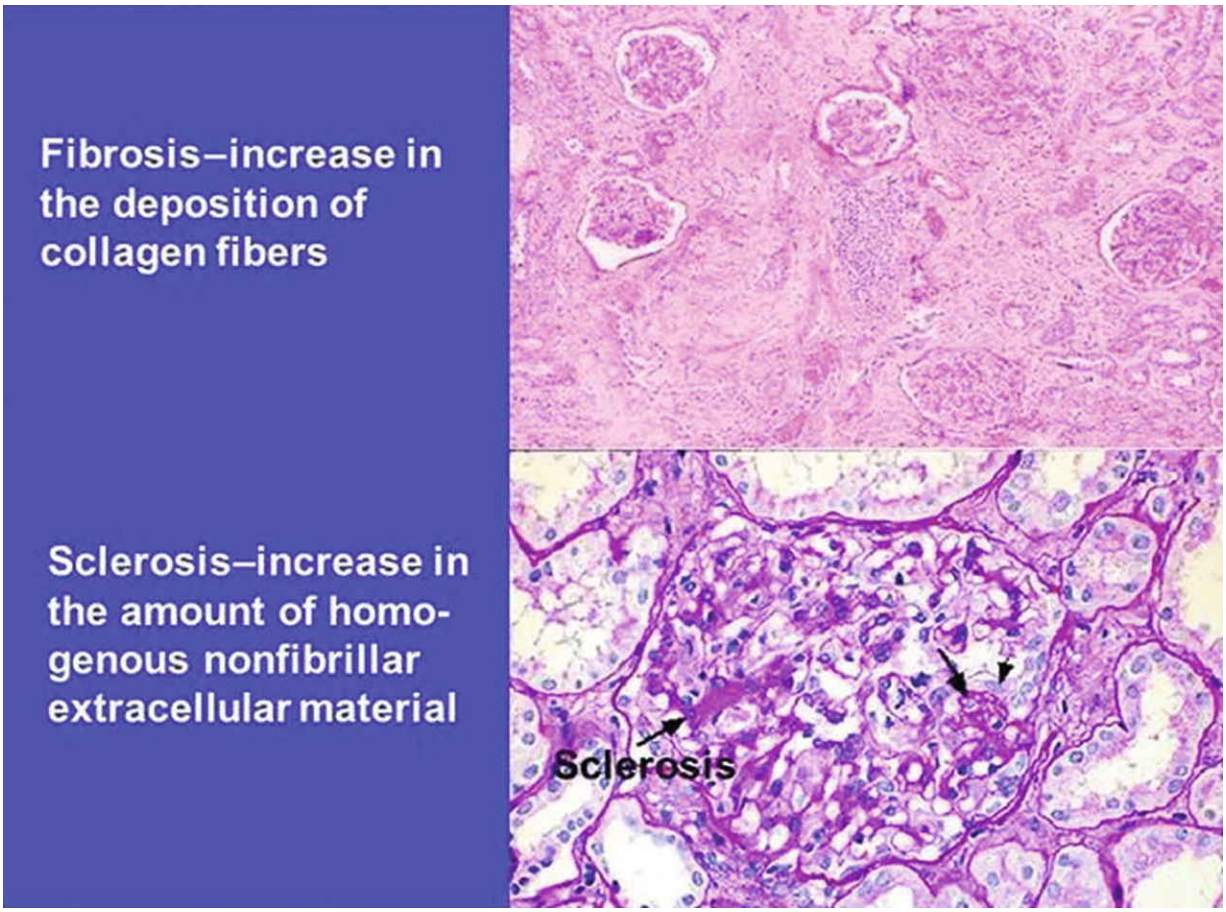


Figure 16-24. Sclerosis/fibrosis. (Sharma, V. *Medizzy*. Available at: https://medizzy.com/feed/4179963. Accessed November 20, 2019.)

- Adipose tissue (Figs. 16-25 and 16-26)

Figure 16-25. Adipose tissue: lymphedema and lipedema. (aliposuctioncenter.com/diagnosing-lipedema/. Accessed November 20, 2019.)

Figure 16-26. Adipose tissue in lymphedema. (*Health Europa*. 2018. Available at: https://www.healtheuropa.eu/a-lesson-on-lipoedema/82966/. Accessed November 20, 2019.)

Clinical Evaluation and Differential Diagnosis

A differential diagnosis is the process by which a provider differentiates between two or more conditions, which share similar symptoms. In the case of lymphedema, a differential diagnosis involves a large amount of background knowledge regarding the pathology and presentation of a variety of diseases and medical conditions. The following is a list of clinical conditions and diagnosis that should be considered with evaluating the patient with lymphedema (see Table 16-1).

Table 16-1 Clinical Conditions and Diagnosis to Consider When Evaluating a Patient with Lymphedema

Clinical Conditions[3,8]	Pathology/Symptoms
CVI (chronic venous insufficiency)	Improper functioning of valves in veins causing swelling and skin changes
DVT (deep vein thrombosis)	A blood clot in the deep venous system usually in legs, commonly presenting with redness, swelling, and tenderness
CHF (congestive heart failure)	Fluid buildup around the heart causing it to pump ineffectively, presents with swelling in legs, ankles, and feet, also presents with rapid or irregular heartbeat
Liver diseases	Can be caused by a variety of factors including infection, drugs/drug use, and genetics, presents with swelling of abdomen and legs, also causes changes in protein levels
Kidney diseases	Diagnosed by a blood test, usually presents with few symptoms, causes fluid and waste build up in the body, can present with hypertension, electrolyte imbalance, swelling, and unintentional weight loss
Malnutrition	Caused by poor diet or protein energy imbalances, presents with metabolic and energy compromise, manifesting weight loss
Lipedema	Condition that causes excess fat to accumulate in the lower part of the body, usually presents in women, no improvement of symptoms with elevation or compression
Myxedema	Severe form of hypothyroidism, causes swelling in face, legs, and tongue, also causes weight gain and dry skin
CRPS (complex regional pain syndrome)	Complex form of regional pain syndrome effects extremities, causes swelling and changes in skin color
Fluid retention syndrome	Also known as idiopathic edema, mainly presents in females, causes bloating, generalized weakness fibromyalgia, cause unknown, edema develops over time in hands, face, and legs
Neurological conditions	Recent studies show a connection to lymphatic system and the brain
Medications	A side effect of many medications is swelling, notable medications are • Calcium channel blockers • NSAIDS • Corticosteroids • Hormone treatments and therapy • Anticonvulsants • Antidepressants • Antidiabetics • Antipsychotics • Chemotherapy agents • Immunosuppressants

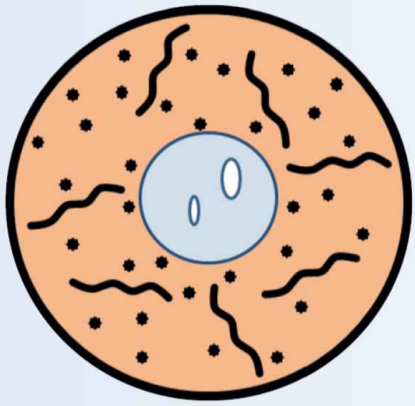
Stages of Lymphedema

In order to develop a treatment protocol, understanding the stage of lymphedema is important. This is done by obtaining a thorough medical history, “edema/lymphedema work-up” and assessing the clinical presentation of the affected area/limb. Lymphedema stages are described below[3,8,12,13,17]:

- **Stage 0 Lymphedema**, also known as subclinical or latency lymphedema is the earliest stage of lymphatic impairment. Though lymph transport is afflicted, there are no signs of edema. The patient may use subjective terms of the limb or area feeling “tight, full, heavy, achy.”
- **Stage 1 Lymphedema**, also called early-onset or reversible lymphedema. Clinical presentation in this stage includes presence of edema and possible pitting, but changes in tissue integrity are minimal. The patient is likely to report subjective complaints of how the affected limb or area “feels.” Swelling often responds to elevation in this stage.
- **Stage 2 Lymphedema** or spontaneously irreversible may be subdivided further into “early and late stage 2.” In early stage 2, pitting edema may still be present but tissue changes are occurring, and the limb is no longer responding to elevation. Later stage 2 demonstrates nonpitting edema with fibrosis. The patient not only has subjective complaints but functional movement in the affected area is decreased and the fit of clothing or shoes becomes tight or unattainable.
- **Stage 3 Lymphedema** has also been called in the past lymphostatic elephantiasis. Prolific skin changes occur in this stage and infections can be frequent. Presentation of the affected area includes deep skin folds, hypertrophy, papillomas, fibrotic tissue throughout, and disfigurement. Functionality is significantly compromised.

Stages of Lymphedema and Treatment (see Table 16-2)

Table 16-2 Stages of Lymphedema and Treatment

Stages of Lymphedema	Development in Epifascial Compartment	Characteristics and Presentation	Treatment
Stage 3 Lymphostatic Elephantiasis	Protein-rich edema with increased epifascial volume, fibrosclerosis, and severe skin-tissue alterations 	• Severe swelling, fat deposition, absence of pitting edema, fibrosis, trophic skin changes (papillomas, deep skinfolds), (+) stemmer. • Disfiguring limb. • Cellulitis is common.	Evaluation and Education: • Infection prevention, risk-reduction practices, home exercise program (HEP), weight management/ nutritional counseling. • Consistent CDT (daily bandaging and MLD 4–6 weeks or greater, depending on severity). • Transition to day and nighttime garments (preferably custom) and instruct on compliancy protocol. • Pneumatic compression pump indicated. • Follow-up visits with CLT minimum of every 6 months unless needed sooner.

MLD, manual lymphatic drainage; CDT, complete decongestive therapy; CLT, certified lymphedema therapist.
Vodderschool.com/manual_lymph_drainage_overview. Accessed Feb 29, 2020. Modified and adapted from JA. Niezgoda, 2019.

Initial Edema/Lymphedema Patient Consultation

The following is a suggested approach to assessing the patient presenting with tissue swelling and is intended to serve as a guideline for the evaluation of the patient with edema and lymphedema. As with any evaluation in medicine, one must begin with a complete and thorough history and physical examination.

History:

- Identify factors and comorbidities causing swelling
- History of cancer, lymph node dissection, radiation

- Surgical history
- Current medications

Physical Exam:

- Heart and lung
- Vascular (venous and arterial)
- Lymph nodes
- Skin including wounds and ulcers
    - Presentation of leg: shape, color, diminished hair growth
    - Skin integrity/Stemmer's sign/pitting edema/ fibrosis

Vascular Interrogation:

- Ankle–brachial indexes
- Venous duplex
- Echocardiography

Laboratory Testing:

- CBC
- Metabolic panel
- Nutritional indices—albumin/prealbumin
- TSH
- BNP

Consider Common Medical Causes of Edema:

- Chronic venous insufficiency (CVI)
- Heart failure/cardiac
- Obesity
- Acute thrombosis
- Limb dependency
- Baker's cyst
- Venous outflow obstruction (any cause)
- Lymphedema: primary or secondary
- Phlebolymphedema (secondary lymphedema)
- Liver disease/cirrhosis
- Nephrotic syndrome
- Calf muscle dysfunction
- Sleep apnea
- Pulmonary hypertension

- Trauma
- Medications that can cause or contribute to edema

Consider Uncommon Medical Causes of Edema:

- Protein losing enteropathy
- Thiamine deficiency
    - Beriberi
- Autoimmune inflammatory disease
    - Polymyalgia rheumatica
    - Scleroderma
- Parvovirus B19
- Hypothyroidism pretibial myxedema

Medications that More Commonly Can Cause or Contribute to Edema:

- NSAIDS (5% risk)
    - Nonselective and selective Cox-2 inhibitors
    - Phenylbutazone
- Cardiac
    - Calcium channel blockers
    - Ace inhibitors (angioedema)
    - Beta blockers (clonidine)
- Direct Vasodilators
    - Hydralazine, minoxidil, methyldopa, olanzapine
- Antisympathetic Agents
    - Guanethidine, reserpine
- Hormones
    - Corticosteroids, estrogens, progesterones, testosterone
- Diabetic
    - Thiazolidinediones, pioglitazone, rosiglitazone

Medications that Uncommonly Can Cause or Contribute to Edema:

- Antibiotics
    - Levofloxacin
- Antidepressants
    - Monoamine oxidase inhibitors
- DMARDs (disease-modifying antirheumatic drugs)
- Endothelin receptor antagonists
    - Ambrisentan, bosentan

- Erythropoietic agents

(Adapted from Wade Farrow, MD's—iCertus Best Practice Algorithm, klosetraining.com/klc-2019/workshop-a)

Collaborative Management of Lymphedema

Lymphedema Therapist

The role of the lymphedema therapist is to develop a plan of care based on medical history, thorough understanding of lymphedema staging, and mixed etiologies. In addition to clinical presentation, it is important to develop an understanding of the patient's personal profile and personal history. The lymphedema therapist examines the patient's functionality, deciphering if the patient will be able to achieve the treatment protocol independently or if additional assistance is required. The plan of care is best achieved when the therapist, physician(s), additional healthcare providers on the team, patient, and family members work together to maximize the patient's potential in independence.

Wound Care Specialist

One of the most common dilemmas facing wound care clinicians is differentiating between venous disease, which is often associated with edema, and other causes of edema and lymphatic disorders. Differentiating between these conditions is important because treatment profiles differ considerably. Managing the patient with a nonhealing wound or ulcer is challenging. When additionally compromised by tissue swelling or lymphedema, efforts to promote are additionally difficult.

Vascular Interventionalist

The clinical linkage of the lymphatic and vascular cannot be overstated. Aggressive vascular evaluation, interrogation, and intervention in patients with tissue edema and nonhealing wound is critical to successful outcomes.

Collaborative Setting and Comprehensive Approach

The management of a patient with edema, lymphedema, nonhealing wounds, arterial, and venous disease commonly requires the skill set and mind set of a team of specialists working in consort. There is frequently overlap in the disease manifestations and commonly these patients all share clinical findings of swelling or edema. Working together and collaborating on the management of these patients will yield improved care and provide superior outcomes.

Lymphedema Treatment

Prior to multilayer bandage application, MLD is performed on the anterior and posterior trunk and left lower extremity (Fig. 16-27). Wound treatment applied prior to application of bandages. Wound care protocols are executed per facility policy. Evaluation of bacterial presence and infection/inflammation is coupled with standard debridement as required. Meticulous attention to providing patient-wound appropriate topicals and dressings is mandatory. Lymphedema treatment should not cause an alteration in standard wound care protocols, can be delivered concurrently, and can provide for enhanced healing outcomes.

Figure 16-27. Lymphedema treatment in the wound care clinic. (AZH Wound and Vascular Center, Milwaukee, Wisconsin; Photo courtesy of J. Niezgoda.)

Series of Treatment Photos (Figs. 16-28 to 16-31)

Figure 16-28. Lymphedema therapy supplies. (AZH Wound and Vascular Center, Milwaukee, Wisconsin; Photo courtesy of J. Niezgoda.)

Figure 16-28 demonstrates the amount and variety of layers required with proper multilayer bandage application for full leg decongestive therapy.

Initial layer for full leg lower extremity: toe wraps, stockinette, cotton and foam padding over dorsal and malleoli surfaces (Fig. 16-29).

Foam padding applied. In cases of higher edema and fibrosis, additional thickness and variety of foams should be used (Fig. 16-30).

Full leg multilayer wrap (Fig. 16-31).

Figure 16-29. Cotton wrap layer. (AZH Wound and Vascular Center, Milwaukee, Wisconsin; Photo courtesy of J. Niezgoda.)

Figure 16-30. Foam layer. (AZH Wound and Vascular Center, Milwaukee, Wisconsin; Photo courtesy of J. Niezgoda.)

Figure 16-31. Full leg wrap. (AZH Wound and Vascular Center, Milwaukee, Wisconsin; Photo courtesy of J. Niezgoda.)

Lymphedema Treatment

The international gold standard of treatment for lymphedema is complete decongestive therapy (CDT). A healthcare practitioner that is certified in lymphedema treatment through the Lymphology Association of North America (LANA) demonstrates the skillset to provide CDT.

“Currently, there is no cure for lymphedema; the main goal of the treatment, therefore, is to return the lymphedema to a stage of latency, utilizing remaining lymph vessels and other lymphatic pathways. The normal or near normal size of the limb should be maintained and reaccumulation of lymph fluid should be prevented. Additional goals are prevention and elimination of infections and the reduction and removal of fibrotic tissues.”[12]

Phases of Lymphedema Therapy

Decongestive/Intensive Phase

The goal in this phase is to reduce limb volume and girth, soften fibrotic tissue, and increase lymphatic load uptake to decrease congestion within the interstitial tissue space. By decreasing limb volume, wounds have increased opportunity to heal. The following provides a sequenced approach for the decongestive phase of therapy:

Manual Lymphatic Drainage (MLD) is the manual treatment approach based on Dr. Emil Vodder's specialized strokes.[1] MLD is different from massage therapy. The MLD approach is quite gentle, providing specific directional pressure in the working phase and releasing pressure in the resting phase to increase lymphangiomotoricity. This specialized manual treatment approach redirects lymph flow to healthier functioning areas. Massage therapy is not recommended in the treatment of lymphedema as the high manual pressure applied to treat muscle tissue, tendons, and ligaments increases vascular flow which, in turn, increases lymph fluid formation.[3,8]

With MLD, the trunk and unaffected area is stimulated first as it can take on the additional fluid. The edema is gently moved across the lymphatic watersheds, toward the healthy lymphatic structures and regional lymph nodes. The affected area is the last to be treated as the optimal movement of lymph fluid is from proximal to distal. Results from MLD include decreased swelling in the congested area, decreased lymph load, decreased fibrosis, increased development of collateral drainage routes, and increase in stimulation of the functional structures within the lymphatic system. The gentle approach of MLD also provides a calming, analgesic effect that can be very soothing to the patient[3] (Fig. 16-32).

Application of Multilayer Compression Bandages includes specific layers of varying widths of cotton, foams (depending on severity of fibrotic tissue), and short stretch bandages. By applying more layers

and stretch at the distal extremity, increased gradient pressure occurs to facilitate lymph movement. The short stretch bandages create a high working pressure and low resting pressure for muscle contraction to increase lymphangiomotoricity. They are used in phase 1 of CDT, and once the maximal reduction in size of limb has been achieved, the patient is transitioned to a maintenance garment.[3,12]

Remedial Exercises and Self-MLD are prescribed by an occupational or physical therapist and are to be performed while wearing the bandages to optimize lymph movement. The goal of these exercises is to increase strength and range of motion, while increasing lymphatic circulation. Self-MLD and diaphragmatic breathing should also be taught during this time for lymphatic stimulation.

Skin/Nail Care and Education are essential in wound and lymphedema treatment. The underlying principle for nail care in patients is prevention. Lymphedema increases the chances for cracks and fissures in the skin, which leave the patient susceptible to infection. Lymph fluid that has built up in the lower legs is very protein rich providing a good breeding ground for bacteria and other microorganisms. For taking care of the skin, the basic principle is moisturizing with appropriate lotions, these are lotions that are hypoallergenic, contain no fragrance, and have a neutral or acidic pH range. Compression stockings can sometimes irritate the more delicate skin of patients with lymphedema; if these problems are encountered, a different material for compression garments should be considered. For nail care, the nails of fingers and toes should be kept short and cut straight across, care should be taken to prevent ingrown nails, and acrylic nails should be avoided.

Self-Care Management, education, and instructions are provided by the patient's wound care and lymphedema treatment team. Follow-up visits with providers as needed. Postdecongestive therapy, recommended follow-up visits vary according to stage and severity of lymphedema. Assuming a flare up of swelling or wound has not occurred, a 3- or 6-month follow-up is sufficient, provided the patient demonstrates a thorough understanding of expectations for maintenance phase. It is not recommended to go longer than 6 months as the maintenance compression garment loses efficacy.[3,8,12]

Figure 16-32. Manual lymphatic drainage. (AZH Wound and Vascular Center, Milwaukee, Wisconsin; Photo courtesy of J. Niezgoda.)

Maintenance/Self-Care Phase

The goal in this phase is to maintain the decongested limb by transitioning the patient to proper fitting daytime and nighttime compression garments. A pneumatic compression pump should also be considered when transitioning the patient to the self-care phase. Exercise, nutrition, proper skin, and nail care are essential.

Self-Care: Once the limb is decongested and the volume and girth measures have plateaued, the patient is transitioned to the maintenance/self-care phase. In order to maintain the size of the decongested limb, the gold standard of care for lymphedema treatment is to convert the compression therapy from short stretch bandages to a daytime and nighttime garment. Self-care education/training and the patient's ability to demonstrate an understanding of skills learned is fundamental for lymphedema and/or wounds not to exacerbate. A comprehensive team approach with wound care, vascular, and the lymphedema therapist is imperative for success.

Daytime and Nighttime Garments that are prescribed depending on stage and severity of lymphedema. There is an increasing number of ready-made garments available on the market, which is cost-saving to the patient. However, depending on the stage and presentation of lymphedema, a custom garment is typically the best option. With a lymphedema diagnosis, wearing compression is lifelong and a comfortable garment will facilitate compliance (Fig. 16-33).

A LANA-Certified Lymphedema Therapist is best trained for determining maintenance garments. Garments prescribed depend on stage and severity of lymphedema. The patient's ability to don/doff compression should also be considered. The continued advancements in compression and assistive devices are increasing the patient's independence in donning/doffing as well as his/her compliancy of wearing garments consistently (Fig. 16-34).

A frequent error made by providers is the lack of prescribing a nighttime garment with lymphedema diagnosis. If cost of the garment is preventing nighttime compression therapy, the multilayer short stretch bandages used in the decongestive/intensive phase may be used. Velcro inelastic garments are growing in popularity as they may be utilized for both day and night garments and are easier in donning/doffing for patient or caregiver. Chipped foam garments are also excellent options as they facilitate stimulation to the lymphatic channels for increased lymph flow and aid in softening fibrotic tissue.

The efficacy in compression garments and short stretch bandages lasts approximately 3 to 6 months. Some flat knit garments used for daytime and nighttime garments (chipped foam and Velcro inelastic) may last longer, but it is important to educate the patient on the potential of swelling flare ups when the therapeutic compression has decreased efficacy[3,8,12] (Fig. 16-35).

Daily exercises are prescribed by an occupational or physical therapist for lymphatic stimulation. Aquatic therapy is an advantageous form of exercise for the lymphatics as the compression rate in water on an immersed body is 22.4 mm Hg/foot; therefore, an individual standing in 48 inches of water is benefiting from 89.6 mm Hg of compression[18] (Fig. 16-36).

Any type of calf pump facilitating exercises while wearing proper fitting compression is advantageous. The patient should also be well versed in diaphragmatic breathing for thoracic duct stimulation as well as self-MLD.

Rebounding with use of trampoline demonstrates a beneficial exercise[19]; in some recent studies, the effects of jumping on a trampoline were studied in respect to a variety of factors, including jump height, BMI, and oxygenation consumption. These parameters were compared between a group of runners and a group of people jumping on trampoline. What was found in this study was that the BMI for the participants marginally decreased over the 6-week training session 0.09% decrease for running group and 0.39% for jumping group, the oxygen consumption increased 3.05% for the running group versus 7.82% for the jumping group. The vertical jump dramatically increased for the jumping group compared to the running group, 0.35% increase for the running group compared to 13.55% increase for the jumping group. In relation to lymphedema, it can be inferred that the extra exercise will help anyone with lymphedema, but the relatively low impact of trampoline exercises is far more beneficial. Also the increase in jumping height can be interpreted as an increase in lower extremity muscle mass, which will allow for better calf pump functioning and increased circulation within the venous and lymphatic system.[19]

Figure 16-33. Compression day/night garments. (L & R, used with permission.)

Figure 16-34. Dawning compression garments. (AZH Wound and Vascular Center, Milwaukee, Wisconsin; Photo courtesy of J. Niezgoda.)

Figure 16-35. Types of compression garments. (L & R, used with permission.)

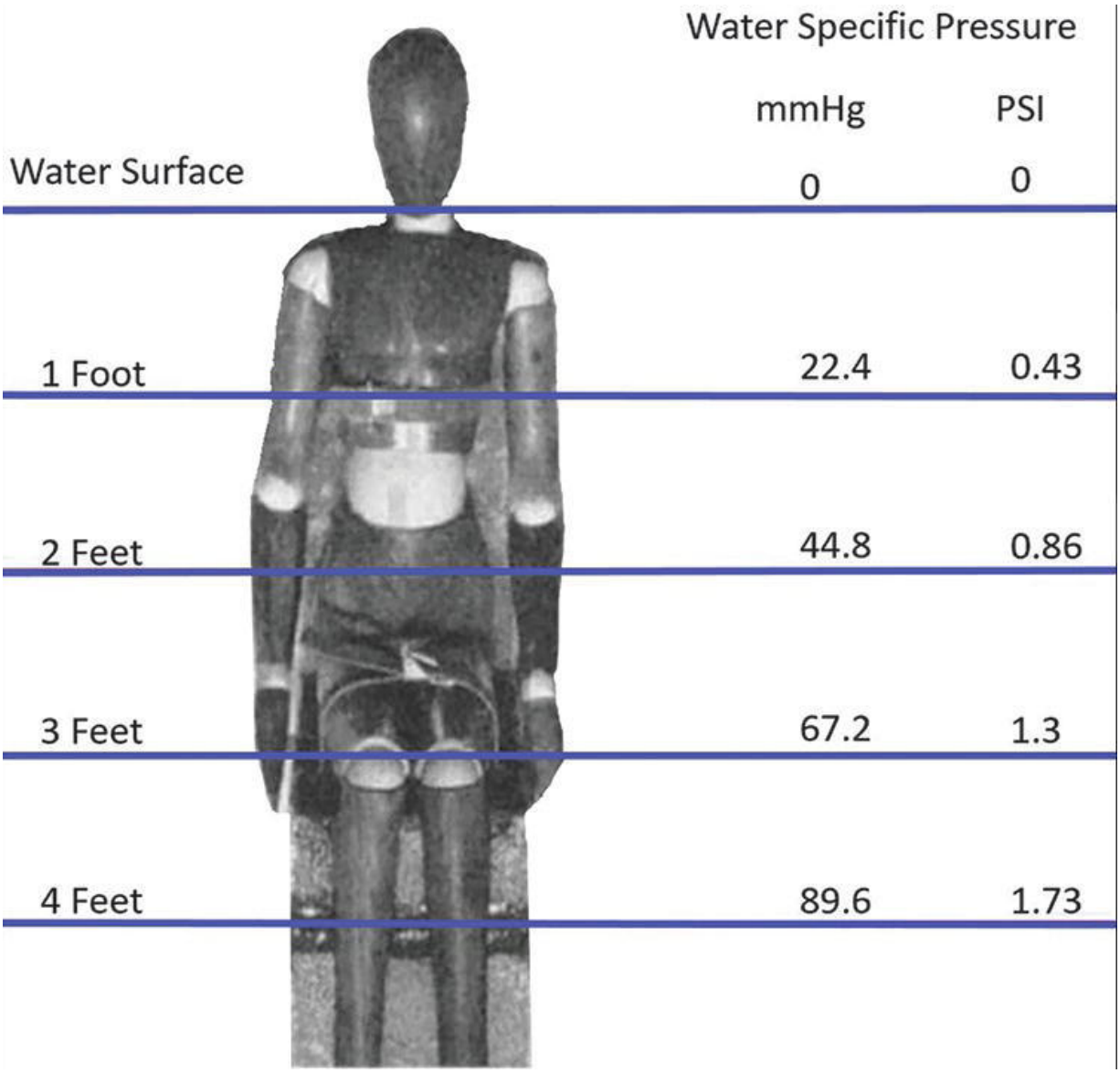


Figure 16-36. Compression of H_2O on body. (https://spacemedicineassociation.org/download/history/history_files_1961/32111031-1.pdf)

Additional Treatment Modalities

Pneumatic Compression Devices

Pneumatic compression devices (PCDs) do not replace MLD in the decongestive phase; however, it is an excellent tool for the patient to use daily during the maintenance phase. The advancement of PCDs not only include extremity sleeves but also include truncal component options, sequential gradient compression with 4 to 8 chambers, and adjustable settings to accommodate the individual's specific needs. Contraindications for use of pneumatic pump include acute infection, acute deep vein thrombosis, untreated CHF, and kidney disease.[3,8] Insurance coverage on pumps vary.

A study was conducted by Desai et al. to compare the clinical use of MLD, compression, and pneumatic therapy.[20] A meta-analysis was also performed comparing more advanced and less advanced pneumatic pumps. In this study, they measured limb volume measures, BMI, a leg lymphedema complexity score, and SF-36 quality of life measurement. They found that with pneumatic compression, there was a 28% decrease in limb volume, a substantial decrease in BMI, a significant increase in SF-36 quality of life measurement in all but one area, and a significant improvement of leg lymphedema complexity score. Also they calculated that per patient, there was a savings of $3,200 in medical expenses relating to lymphedema. The meta-analysis found that the advanced 8 chamber pneumatic pump accounted for more pronounced and faster reduction in limb volume, the basic pump decreased limb volume by 21.8% in 1 year, whereas the advanced pump decreased limb volume by 31.2% at 1 year. The average BMI (n = 128) was 36.4, 3 months in it was 35.1 and at 1 year it was 34.0. This study suggests that a pneumatic pump can be a very good supplement to a lymphedema therapist's toolbox[20] (Fig. 16-37).

Figure 16-37. Pneumatic pump compression. (http://acutewoundcare.com/index.php/products/compression-pumps)

Kinesiology Taping

Kinesiology taping for lymphatic drainage and fibrotic or scar tissue has demonstrated value as an adjunct treatment for lymphedema therapy (Fig. 16-38).

Figure 16-38. Kinesio taping. (AZH Wound and Vascular Center, Milwaukee, Wisconsin; Photo courtesy of J. Niezgoda.)

Medications

Diuretics are commonly used in health care and they are utilized to management a variety of medical conditions, some of these have edema as a symptoms or clinical finding. However, for the patient with a diagnosis lymphedema, diuretics are NOT beneficial and in fact cause more damage to the lymphatics. No medication has been demonstrated to be curative for lymphedema.

Low-Level Laser Therapy

Low-level laser therapy is a treatment that uses a low-level infrared or near-infrared laser light to reduce inflammation and can help with the

reduction of scar tissue as well as fibrotic tissue. This modality of treatment has been mainly utilized with breast cancer–related lymphedema, and currently there are very few clinical trials but this is showing very promising results.[21]

Nutritional Therapy

Nutritional therapy is an important component to be considered when working with patients with lymphedema as obesity further exacerbates lymphatic damage. Nutritional counseling referral should be determined when assessing BMI.

Patient Education

Patient education is imperative to facilitate compliance and success with a lifelong disease process. It is important to armor the patient with a knowledge base on the lymphatic system, where the compromise is occurring in his/her body, and the best individualized treatment practice during the active and maintenance phases.

Venous Intervention

The management of venous insufficiency may be a beneficial adjunct to the treatment of lymphedema.

Subfascial endoscopic perforator surgery (SEPS) is a procedure that ligates any abnormal perforator veins in the legs. A surgical clip is placed on each perforator needing it through a tiny incision in the leg. This procedure takes about 3 hours and can be done under local or general anesthesia.

EVLA or endovenous laser ablation is a procedure where a provider inserts an optical laser catheter into a vein usually the great saphenous vein or lesser saphenous vein. The catheter then uses laser energy to cauterize the vein closed and prevent further blood from pooling in that specific vein. This procedure is usually done under local anesthesia and the patient can leave the same day.

RFA or radiofrequency ablation is similar to EVLA, but instead of a laser catheter, it is a radiofrequency catheter, which uses radio waves to heat up a heating element in the catheter and cauterize the vein.

This procedure is slightly more invasive because the tissue surrounding the vein needs to be anesthetized prior to cauterization of the vein.

Sclerotherapy can be separated into two different types of procedures, perforators, and superficial venous treatments. Perforator sclerotherapy is a type of sclerotherapy where smaller perforator veins closer to the skin are injected with a sclerosant medication that is mixed with saline until it is a foam-like consistency. This procedure is limited by dose so patients with many perforators may need a few treatments to completely kill the veins. Superficial venous treatments are a recent development for treating the greater saphenous and lesser saphenous veins. A type of sclerosant medication is mixed with an oxygen–carbon dioxide gas to create a foam, which is then injected into the GSV and SSV or other incompetent vein. This procedure is minimally invasive and does not require anesthetizing the entire vein, only the dermis at the injection site. The patient can leave the same day and procedure only takes about 30 minutes.

Contraindications for Lymphatic Treatment

Contraindications for lymphatic treatment include diagnosis of acute deep vein thrombosis, acute infection, and untreated CHF and kidney disease. Compression is warranted with acute DVT and is not contraindicated. Malignancy and renal dysfunction may be indicated for lymphatic treatment; however, a physician's prescription is required. Close communication between referring provider and treating lymphedema therapist is recommended.[12,13]

Imaging Technologies

The ability to view the underlying fluid transport structures has been a dream of many providers working in wound care, vascular surgery, and lymphedema. Until recently, the only option was to use radiographic imaging techniques, x-ray, CT, MRI, and others.[16,19] These all have limitations, but the major one is exposing the patient and the providers to radiation. These types of imaging include lymphoscintigraphy, which is mainly used for cancer mapping.[22] In this procedure, tiny radioactive particles are injected into the primary cancer site. A gamma camera is used to track these molecules as they are being transported through the involved tissues.[22] Another option is lymphangiography, which is the injection of a radiopaque substance into the lymph system, which is then imaged radiographically. Both of these studies expose the patient to radiation and result in very crude images with limited clinical utility.[16]

A more recent technology has developed for noninvasive tissue imaging, which may be a feasible option for imaging edema and lymphedema without the drawbacks of harmful radiation. Near-infrared spectroscopy uses near-infrared light to visualize the underlying blood oxygenation levels in the tissues. A variant of this technology was produced that is able to measure and visualize the amount of interstitial fluid in a given area. This technology is not very widely known and will require additional research to provide a devise that is usable in a clinical setting. It is hoped that this technology will be able to visualize very concentrated edema that is created from a very local histamine response. Near-infrared cameras are available now, but these cameras measure the difference in deoxygenated to oxygenated hemoglobin and are more specifically made for wound care and surgical procedures.[4,13]

Obesity and Lymphedema

Although the pathophysiology of lymphedema is unknown, the disease is characterized histologically by fibrosis and abnormal adipose deposition. Clinical studies have provided evidence that obesity and postoperative weight gain are significant risk factors for the development of lymphedema. A study by Mehrara et al. has shown that extreme obesity can result in markedly impaired lymphatic function and primary lymphedema.[23]

Summary

Lymphedema results when there is an imbalance between capillary filtration, lymphatic drainage, resulting in impairment to the lymphatics. Lymphedema occurs most commonly due to injury or obstruction, such as from cancer treatment, or associated with severe venous insufficiency and or obesity.[23] Clinical judgment based on findings from a comprehensive assessment and patient history should guide the treatment decision. The earlier that MLD and compression therapy can be initiated, the better the outcome will be for the patient. Compression therapy is an active therapy that is generally underused. But when used on the right patient in the right way so that concordance is maintained, it is the key to improving patient outcomes. The care and management of lymphedema patients is usually followed-up in outpatient centers or wound care clinics.[24,25] Experts representing vascular surgery and orthopaedic surgery, as well as the disciplines of plastic surgery, dermatology, endocrinology, physical therapy and wound care specialists, and nursing, collaborate to provide a comprehensive approach to wound healing, limb preservation, and follow-up care. There is no cure for lymphedema, but treatment with lymphatic compression, qualified practitioners, and a long-term plan can effect significant improvement and control of symptoms, which may include skin changes, limb distortion, and cellulitis (LE&RN and Wound Management Textbook).[11]

PATIENT SCENARIO

Clinical Data

Case 1 (Fig. 16-39)

Figure 16-39. Case Study 1. (AZH Wound and Vascular Center, Milwaukee, Wisconsin; Photo courtesy of J. Niezgoda.)

This patient is a 58-year-old male with medical history of chronic venous insufficiency, lymphedema, oral pharyngeal cancer, transverse myelitis and past surgical history of right total hip replacement, cervical fusion, and treatment for oral pharyngeal cancer. Patient experienced increased bilateral lower extremity swelling (greater in the left lower extremity) affecting functional mobility. He was referred for lymphedema treatment prior to vascular treatments to decrease bilateral lower extremity swelling (optimize for vascular treatment). Complete decongestive treatment was facilitated

to the left leg first as it was the more problematic limb. Decongestion consisted of daily manual lymphatic drainage and application of multilayer short stretch bandages. Patient was provided a home exercise program for lymphatic stimulation and education on skin and nail care. During phase 1 of decongestive therapy of the left lower extremity, edema in the right lower extremity reduced as well. After 1 week of decongestive therapy for left lower extremity, the following girth measurements were obtained:

Initial Evaluation	1 week after Initial Evaluation
R/L in cm:	R/L in cm:
Foot Arch: 28.7/28.2	26.8/26.0
Malleoli (Diagonal Heel Measure): 38.2/39	36.7/35.5
Ankle: 30.7/30.6	27.9/27.6
10 cm Superior to Ankle: 35.6/39.5	32.7/34
Calf: 50.7/53.3	45.2/45.4
Patella: 46.5/48.3	46.5/45.7
Thigh: 71/70.5	67.5/64

Due to increased fibrosis in the left lower extremity, patient was transitioned to a nighttime chipped foam short stretch garment with zipper and Velcro closures and a 30 to 40 mm Hg flat knit knee high garment for daytime. The right lower extremity was also transitioned to inelastic short stretch Velcro garment for nighttime and 30 to 40

mm Hg flat knit garment for daytime post complete decongestion therapy. The notable measurements within in the 1-week timeframe of decongestion included right ankle girth decrease of 4 cm, right calf girth decrease of 7.7 cm, left ankle girth decrease of 2.9 cm, and left calf girth decrease of 9 cm. Following lymphedema treatment, patient was then well managed with superficial endovenous ablation for his underlying venous insufficiency.

PATIENT SCENARIO

Clinical Data

Case 2 (Fig. 16-40)

Figure 16-40. Case Study 2. (AZH Wound and Vascular Center, Milwaukee, Wisconsin; Photo courtesy of J. Niezgoda.)

This 60-year-old nondiabetic female with CVI, ulceration, and lymphedema presented to clinic with bilateral lower extremity venous ulcers. She was evaluated and treated by both wound care and the lymphedema specialist. Wound treatment included topical antimicrobial and compression. Lymphedema treatment was started on the left lower extremity first due to severity of wound and affected functional movement. Therapy included decongestive phase of manual lymphatic drainage, application of multiple layers of short stretch bandages, home exercise program, kinesiology taping for stimulation of lymphatic drainage, and skin/nail education. The left leg ulcer healed, and limb was decongested within 2 weeks. The patient was then transitioned to an inelastic, short stretch Velcro garment on the left lower extremity, which was used 24/7. The right lower extremity was then treated with the same lymphedema protocol and limb was decongested within 10 days and transitioned to maintenance inelastic, short stretch Velcro garment, also worn 24/7. During decongestive phase of LLE, the RLE began reducing (without compression) and right leg ulcer demonstrated enhanced healing trajectory as well. The patient's right ankle girth decreased from 40 to 28.3 cm; right calf girth decreased from 67 to 50.5 cm; left ankle girth decreased from 41 to 30 cm; and left calf girth decreased from 67.5 to 52.5 cm. It should be noted that patient was not taking water pills, complete decongestive therapy (CDT) effectively reduced limbs.

Upon initial consult, patient weighed 395 pounds; after 4 weeks of treatment, patient weighed 348 pounds.

Show What You Know

1. **What is considered the international gold standard of treatment for lymphedema?**
    A. ECT
    B. CDT
    C. CVI
    D. DVT

2. **A graded circular knit compression stocking may be prescribed to wear at night as it is a safe form of compression on the lymphatic system, during sleep.**
    A. True
    B. False

3. **How many stages are there of lymphedema?**
    A. 1
    B. 2
    C. 3
    D. 4.

Answers to these questions are found on page 685.

References

1. Vodder School Manual Lymph Drainage History (MLD). Available at: www.vodderschool.com/manual_lymph_drainage_history. Accessed November 25, 2019.
2. Földi, M., Földi E., et al. Földi's Textbook of Lymphology: for Physicians and Lymphedema Therapists, 3rd ed. München, Germany: Elsevier Urban & Fischer, 2006.
3. Klose, G. Klose Training, Lymphedema Certification Course, May 2016. Lafayette, CO.
4. North America Lymphedema Education Association (NALEA). Available at: http://www.lymphedemaeducationassociation.org/about.html
5. Lymphology Association of North America (LANA). Available at: https://www.clt-lana.org/mission- statement.html
6. National Lymphedema Network (NLN). Available at: https://lymphnet.org/
7. Thiadens, S. Available at: https://klosetraining.com /2017/05/31/lymphedema-community-honors-saskia -thiadens/
8. Academy of Lymphatic Studies, Lymphedema Association of North America (LANA) Preparation course, May 2016, Sebastian, FL. May 15-16, Palm Beach, FL, 2016.
9. Bailey, R. Anatomy and Physiology of Lymph Nodes: Lymph Nodes Are More Than Bumps Under Your Skin. Thought Co. September 12, 2018. Available at: https://www.thoughtco.com/lymph-nodes-anatomy-373244#. Accessed November 25, 2019.
10. Lee, B., Rokson, S.G., Bergan, J., eds. Lymphedema: A Concise Compendium of Theory and Practice, 2nd ed. New York, NY: Springer International Publications, 2019.
11. Boundless. Boundless Anatomy and Physiology. Lymphatic Tissue Development & Lymphatic Vessels. Lumen Learning. Available at: https://courses.lumenlearning.com/boundless-ap/chapter/development-of-lymphatic-tissues/vessels. Accessed December 29, 2019.
12. Zuther, J.E., Norton, S. Lymphedema Management: the Comprehensive Guide for Practitioners, 3rd ed. New York, NY: Thieme, 2018.
13. Johnson, J., Yates, S., Burgess, J. Venous Insufficiency, Venous Ulcers & Lymphedema. In: D.B. Doughty, L.L. McNichol, eds. Wound, Ostomy, and Continence Nurses Society Core Curriculum. Philadelphia, PA: Wolters Kluwer, 2016:398-419, Chap 21.
14. Levick, J.R. Revision of the Starling Principle: New Views of Tissue Fluid Balance. The Journal of Physiology 557(3):704, 2004. doi: 10.1113/jphysiol.2004.066118.
15. National Organization for Rare Disorders. Available at: www.nidcd.nih.gov/directory/national-organization-rare-disorders-nord. Accessed December 28, 2019.

16. Warren, L.E.G., Miller, C.L., Horick, N., et al. The Impact of Radiation Therapy on the Risk of Lymphedema after Treatment for Breast Cancer: A Prospective Cohort Study. International Journal of Radiation Oncology, Biology, Physics 88(3):565-71, March 2014.
17. Weissleder, H., Baumeister Rudiger, G.H. Lymphedema Diagnosis and Therapy, 4th ed. Essen, Germany: Verl, 2008.
18. Beckman, E.L., Coburn, K.R. Some Physiological Changes Observed in Human Subjects during Zero G Simulation by Immersion in Water up to Neck. Naval Air Development Center, Johnsville, PA. Rept. NO NADC 6107.AD 256 727, 1961.
19. Witassek, C., Nitzsche, N., Schulz, H. The Effect of Several Weeks of Training with Minitrampolines on Jump Performance, Trunk Strength and Endurance Performance. Deutsche Zeitschrift fur Sportmedizin 2018(2):38-44, 2018. doi: 10.5960/dzsm.2018.318.
20. Desai, S.S., Shao, M. Superior Clinical, Quality of Life, Functional, and Health Economic Outcomes with Pneumatic Compression Therapy for Lymphedema. Annals of Vascular Surgery 2019. doi.org/10.1016/j.avsg.2019.08.091.
21. Cupolo, J., Smoot, B. Low-Level Laser Therapy May Reduce Limb Volume and Pain in Patients with Breast Cancer–Related Lymphedema. Low-Level Laser Therapy May Reduce Limb Volume and Pain in Patients with Breast Cancer–Related Lymphedema—The American Society of Clinical Oncology (ASCO) Post. 2015. Available at: https://ascopost.com/news/21623. Accessed December 9, 2019.
22. Moncayo, V.M., Aarsvoid, J.N., Alazraki, N.P. Lymphoscintigraphy and Sentinel Nodes. Journal of Nuclear Medicine 56(8):901-7, June 1, 2015.
23. Mehrara, B.J., Greene, A.K. Lymphedema and Obesity: Is There a Link? Plastic and Reconstructive 134(1):154e-60e, July 2014.
24. Ehmann, S. Incorporating Specialized Lymphedema Therapy & the Wound Clinic. Today's Wound Clinic 9(6), August 19, 2015. HMP Communications, Malvern, PA.
25. Hettrick, H. Tearing Down the Silos of Lymphedema Care in the Wound Clinic. Today's Wound Clinic 11(2), December 2017. HMP Communications, Malvern, PA.

17

Arterial Ulcers

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We gratefully acknowledge the contributions of Ronald A. Kline, MD, for his work on previous editions of this chapter.

Objectives

After completing this chapter, you will be able to:

- identify the structure and explain the function of the lower extremity arterial system
- assess the signs and symptoms of lower extremity arterial disease and ulcers
- select appropriate vascular laboratory diagnostic testing for lower extremity arterial disease
- evaluate medical and surgical treatment options for lower extremity arterial disease
- design appropriate patient education for prevention and appropriate lifestyle change.

Scope of the Problem

The designation, peripheral vascular disease, commonly refers to arterial problems in the legs. The reader should be aware that some authors also include diseases of the venous and lymphatic systems in their definition of peripheral vascular disease.

Leg and foot ulcers may have several different etiologies, including arterial, venous, and lymphatic disease along with trauma, infections, inflammatory diseases, and malignancy. This chapter describes the arterial component, including anatomy and physiology, and examines the treatment of lower extremity arterial ulcers.

Approximately 8% to 10% of patients in some series with leg and foot ulcers have pure arterial insufficiency.[1] It's estimated that between 1% and 22% of the population over age 60 suffers from lower extremity skin ulcers.[2–5] One patient survey suggested the current estimate may be low because high numbers of patients indicated that they cared for their own ulcers without consulting a healthcare provider.[6] The principal etiology of leg ulcers is chronic venous disease, whereas foot ulcers are much more commonly caused by arterial disease.[1,7–9] Peripheral arterial ischemia is also the most common vascular consult for limited ability to walk.[7] Although chronic wounds have physical, financial, and psychological effects, it's difficult to measure this impact on a patient's activities of everyday living.[10] It's also difficult to obtain accurate etiological information about leg ulcers because no ulcer etiology documentation exists in about one-third of medical records.

A study by Forssgren and Nelzen in Skaraborg County, Sweden,[11] examined the incidence of leg ulcers before and after a targeted leg ulcer prevention program (starting in 1998) aimed at early detection of venous and arterial disease. In 2002, 621 patients with leg ulcers were identified through a cross-sectional population survey. Half of the registered patients were randomly selected and offered clinical examination. A total of 198 patients with 246 ulcerated legs underwent examination and were categorized in detail according to etiology. Venous incompetence was present in 140 (57%) legs and the dominating cause in 94 (38%) of the leg ulcers, 40 (16%) due to deep

venous incompetence. Arterial insufficiency was identified in 90 (37%) legs and the dominating etiological factor in 41 legs (17%), 8 (3%) being critical ischemic ulcers. This intervention reduced the relative risk (RR) of developing a leg ulcer in 2002 versus 1988 to 0.77. The RR of a venous ulcer was reduced by 46% and arterial by 28%, while there was an increase in diabetic ulcers by 29% and multifactorial ulcers by 42%. The targeted intervention to detect early vascular disease reduced the incidence of leg ulcers overall but also decreased the frequency of the remaining ulcers with an arterial or venous etiology.[11]

Vascular Anatomy and Physiology

Vascular anatomy includes the arterial, venous, and lymphatic systems. Vascular ulcers may develop in any of these systems from a variety of causes. For the purposes of this chapter, the discussion is focused on the arterial system.

Arterial System

Lower extremity arterial perfusion begins with adequate cardiac performance. As blood exits the left ventricle, it begins its downward course through the descending thoracic aorta meeting several arterial branches. The intercostal arteries, which arise from the descending thoracic aorta, are the first important collaterals to perfusion in the legs. These become important when they are the sole collaterals in distal aortic occlusive disease. As the aorta exits the thorax and enters the true abdominal cavity, its caliber begins to decrease after every major arterial branch. Its greatest reduction in size occurs distal to the renal arteries.

Lumbar arteries usually arise as paired vessels at each vertebral level in the abdomen. The lumbar arteries become important collateral pathways to the lower extremities in distal aortic occlusions or severe aortoiliac occlusive disease. At the level of the umbilicus, the abdominal aorta bifurcates into the common iliac arteries that in turn branch into internal and external iliac arteries. The internal iliac arteries perfuse the lower sigmoid colon and rectum. They also, by way of the gluteal and pudendal branches, provide another collateral pathway to perfusion of the legs. The external iliac artery becomes the common femoral artery at the level of the inguinal ligament. It’s at this level that one can first appreciate the quality of the pulse wave by palpating the femoral artery.

Aspects of the Femoral Artery

The common femoral artery bifurcates into the superficial femoral artery and the deep femoral artery. The deep femoral artery is the single most important collateral pathway for perfusion of the lower portion of the leg. Its muscular perforators allow reconstitution of the popliteal artery in superficial femoral artery occlusions. The superficial femoral artery becomes the popliteal artery after it exits the adductor hiatus, also known as *Hunter's canal* (Fig. 17-1).

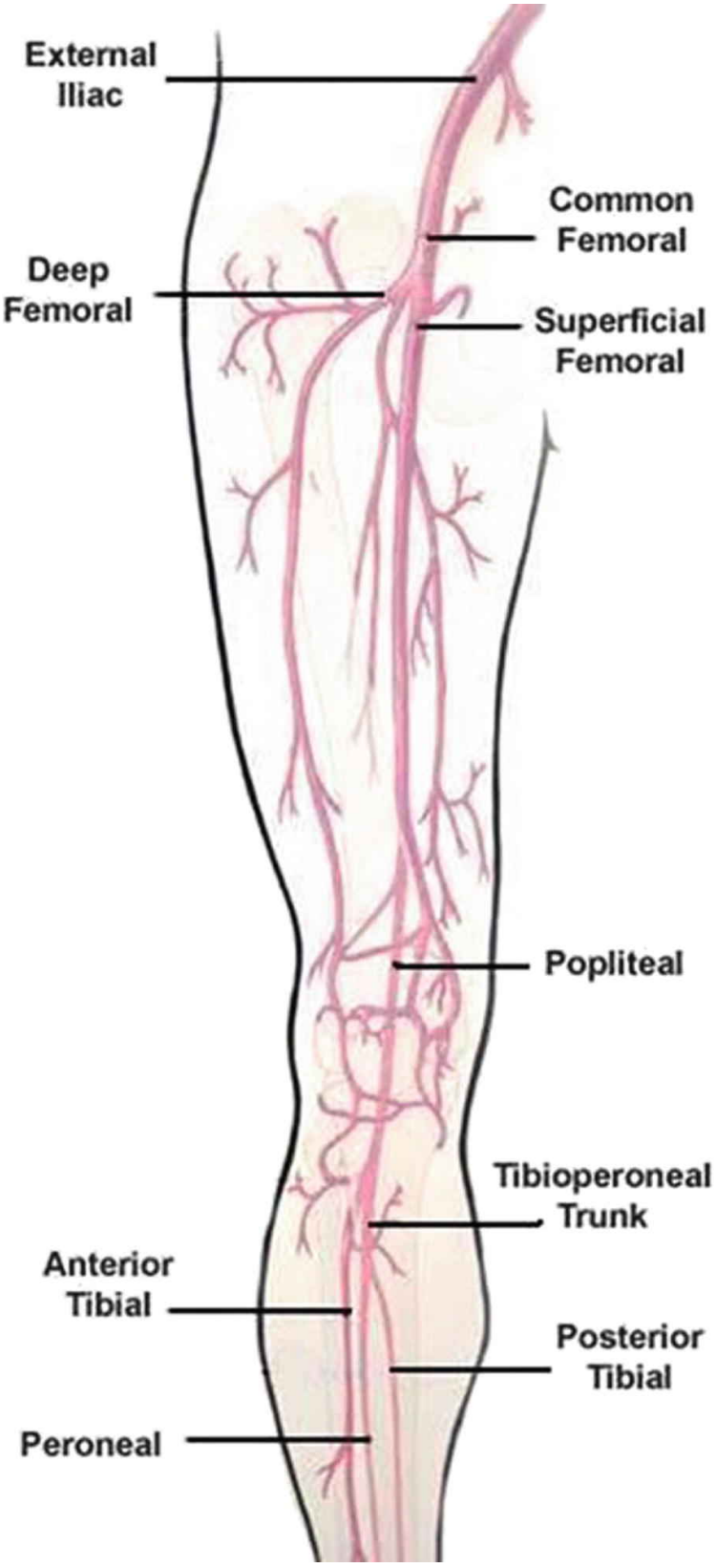
External Iliac
Common Femoral
Deep Femoral
Superficial Femoral
Popliteal
Tibioperoneal Trunk
Anterior Tibial
Posterior Tibial
Peroneal

Figure 17-1. Arterial system. This illustration shows the major arteries of the lower extremities. (Photo courtesy of J. Niezgoda.)

The superficial femoral artery is the most commonly occluded artery in the legs of patients with peripheral vascular occlusive disease. Its occlusion infrequently results in significant ischemia to the lower leg. Below the knee, the popliteal artery bifurcates into the tibioperoneal trunk and the anterior tibial artery. The anterior tibial artery proceeds from the popliteal fossa through the interosseous membrane, which connects the tibia and fibula; it then courses down the anterior muscle compartment into the foot. The tibioperoneal trunk (also known as the tibiofibular trunk) at a variable distance then bifurcates into the peroneal (also known as the fibular) artery and the posterior tibial (PT) artery.

The peroneal artery courses down toward the ankle in the deep muscular compartment, whereas the PT artery descends into the foot in a more superficial fashion. The peroneal artery provides important muscular perforating branches. It's commonly patent even in the presence of severe lower extremity peripheral vascular occlusive disease.

Aspects of the Tibial Arteries

The anterior and PT arteries proceed into the foot with the anterior tibial artery becoming palpable as it becomes the dorsalis pedis (DP) artery. The PT artery then courses behind the medial malleolus and at this level also becomes palpable. The PT artery provides both deep and superficial components to the plantar arch. Perforators from the plantar arch provide arterial perfusion to the heel, sole, and branches to the digits.

The anterior tibial artery, which becomes the dorsal pedal artery and is palpable on the dorsum of the foot, eventually communicates with the plantar arch, forming a complete circuit in the foot. The peroneal artery, although it stops above the level of the ankle joint, does provide medial and lateral tarsal branches that communicate with the distal-most portions of the anterior and PT arteries. This is another important collateral pathway for revascularization of the plantar arch in patients with occlusive disease. Vascular surgeons can perform bypass operations to any of these named vessels, with modern procedures successfully bypassing more distal vessels.

Arterial Wall Architecture

The arterial wall typically consists of three laminae. The outer lamina, the adventitia, is a layer of loose connective tissue that provides moderate strength to the arterial wall. The media, or middle layer, contains both elastic and muscular fibers and is responsible for arterial strength, elasticity, and contractility. The intima, the innermost layer, is the endothelial lining of an artery and a few cell layers thick. As the arterial tree descends from the heart to the periphery, muscular functions become more evident. Vessels below the common femoral artery have a greater propensity for rapid vasoconstriction or vasodilation in direct relationship to perfusion. The tibioperoneal vessels can quickly accommodate changes in perfusion by relaxation or dilation.

Arteries are capable of increasing in size (vasodilation) to maintain constant shear stress when atherosclerotic accumulation decreases luminal surface area. However, once a stenosis reaches 50% of the vessel diameter, the artery loses its ability to dilate any further, and any increase in atherosclerotic accumulation impedes arterial perfusion. Further restriction in flow through this stenotic area results in a decrease in the diameter of the artery distal to the stenosis in order to accommodate diminished blood flow, although at times, due to the increased velocity in the stenotic region, and the increased shear induced stresses, there may be poststenotic dilatation over time. Compliance of an artery decreases as the arterial wall becomes more rigid with calcific atherosclerosis (Fig. 17-2).

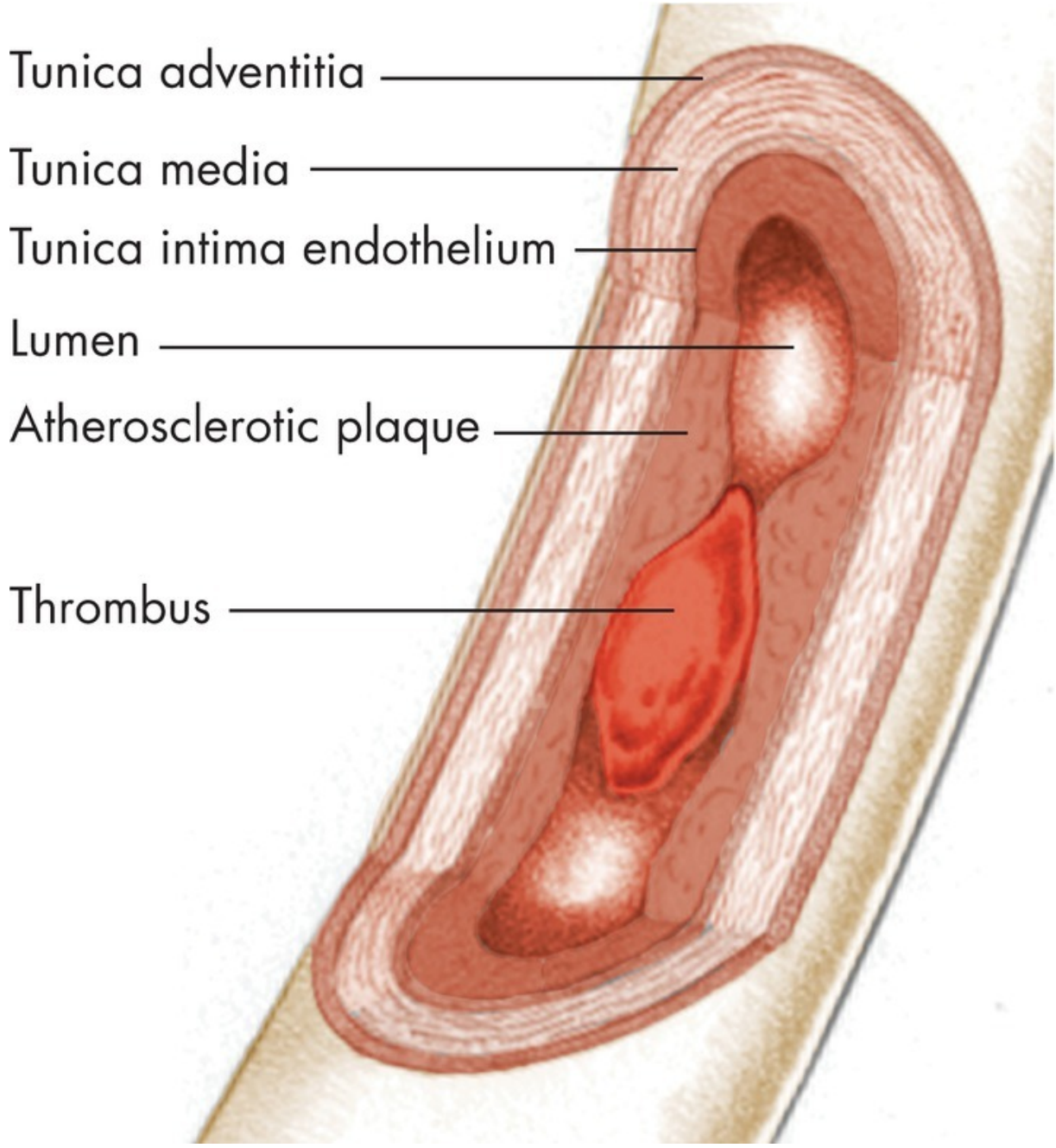

Figure 17-2. Arterial wall. In the layers of the arterial wall shown here, the plaque formation and thrombus significantly reduce blood flow through the vessel.

Arterial Perfusion

As blood descends through successively smaller arterial conduits, it eventually reaches the arteriolar level. Blood flow (rheological factors) in this precapillary bed plays an important role in perfusion. Blood is a non-Newtonian thixotropic fluid (thick or stable at rest but becoming more fluid with motion). The viscosity of blood is inversely proportional to its shear rate or the velocity of blood flow. The slower the blood is propulsed, the more viscous it becomes. The primary determinant of whole blood viscosity at any given shear rate is the hematocrit. As red cell mass increases, blood viscosity markedly increases and the flow decreases.

Dehydration and polycythemia (increased number of red blood cells) are two examples of many disease states that increase whole blood viscosity and can result in blood sludging (sticking together of the red blood cells) in the precapillary bed and a resultant decrease in arterial tissue perfusion. In many elderly patients with arterial occlusive disease, even mild dehydration can result in poor extremity perfusion. Simple rehydration can reduce the red cell mass and allow for better perfusion. In other cases of increased blood viscosity, such as multiple myeloma, plasmapheresis may be necessary to remove the abnormal concentrations of proteins. Nonetheless, in the “normal” atherosclerotic patient, it’s the red cell mass, measured by hematocrit, that is the primary determinant of viscosity.

As blood proceeds into the capillary bed, the diameter of the vessel approaches that of the red cell—approximately 8 μm (microns) in diameter. Red cells pass through capillaries sequentially, improving perfusion at this level. In conditions in which the red cell membrane is relatively rigid, tissue perfusion decreases because of increased transit time for a red cell to pass from the precapillary to the postcapillary level. Although nutrient and oxygen extraction are increased by this increase in transit time, the per-unit perfusion of the tissues is decreased overall. Medications such as pentoxifylline may facilitate red cell deformability, thereby potentially increasing the per-unit tissue perfusion.[7,8] Unfortunately, pentoxifylline has limited efficacy in arterial disease, but cilostazol (inhibiting platelet aggregation and an arterial

vasodilator) improves both pain-free and maximal treadmill walking distance.[12]

In normal states, arterial tissue perfusion is well above minimal requirements, but certain tissues, such as muscle, can change their metabolic requirements. Muscle becomes more efficient under anaerobic conditions (this is called the Cori cycle)—for example, a trained long-distance runner. The process of improving efficiency is gradual, but training to increase muscle capacity is useful in patients with claudication. A regular exercise program can increase the distance walked before claudication occurs. The skin does not have the same kind of compensatory mechanism where exercise can gradually increase blood flow.

Arterial Ulcer Pathophysiology

The pathophysiology of vascular ulcers varies according to the type of ulcer. Arterial ulcers (Fig. 17-3) are wounds that will not heal due to compromised or inadequate arterial blood flow or ischemia. Hypoxia due to anemia can exacerbate ischemia. Precipitating events for arterial ulcers vary. Limbs with arterial compromise may have minimal but adequate blood flow to maintain tissue viability. Ischemic lower-extremity ulcers are often precipitated by trauma or infection.[13]

Figure 17-3. Arterial ulcer on the foot. (Photo courtesy of M. Sieggreen.)

The location of traumatic ulcers varies depending on the cause and site of injury, but these wounds are commonly found on the foot or on the anterior tibial area of the lower leg. Traumatic ulcers may be caused by an acute physical injury, such as blunt trauma (e.g., bumping into a piece of furniture or dropping a heavy object on the foot), or by acute or chronic pressure (such as the continual pressure from ill-fitting footwear). Several other conditions may be responsible for tissue breakdown, including thermal extremes, chemicals, or a localized clot or embolus that can also lead to decreased cellular nutrition from impaired arterial flow. *Regardless of the cause, when ischemia is present, wound healing is inhibited*. It is also postulated that the ischemic–reperfusion injury cycle may be more detrimental to ulcer healing than previously suspected and may be even more damaging than prolonged ischemia.[14] Although some wounds heal in the presence of ischemia, arterial inflow must usually be improved for healing to occur.[15,16] Injury repair requires more than baseline oxygen consumption and increased tissue nutritional need. Diminished arterial flow causes tissue hypoxia, and with injury, this will further aggravate the arterial insufficiency and can eventually lead to gangrene or tissue necrosis (Fig. 17-4).

Figure 17-4. Ischemic forefoot. (Photo courtesy of J. Niezgoda.)

Diagnosing Vascular Ulcers

Vascular disease and ulcer etiology can be determined by obtaining a thorough patient history and performing a physical examination. A focused vascular history includes a clear description of the presenting complaint, past medical history for vascular and related conditions, current and previous medications, and risk factors. Symptoms and signs of lower-extremity vascular disease may include pain or loss of protective sensation, tissue compromise, or change in appearance. Noninvasive vascular laboratory testing is required to identify the location of vascular pathology.

The first question to ask every patient is about his or her history of allergies. This question is important if there is a known sensitivity to medications that may be ordered or to dyes used for computed tomographic angiography (CTA) or conventional angiography. The next question should be about the patient's medications and then about his or her occupation.[15]

The important points to remember about a patient's history include remembering the ABCDES,[15] all of which are increased risk factors for arterial disease. They are:

- **A**$_{1C}$: Hemoglobin A$_{1C}$ detects the average blood sugar over a period of 90 days. Values below 6% are normal, those between 6.1% and 6.5% represent prediabetes, and those above 6.5% represent diabetes. The higher the HbA$_{1C}$, the greater the incidence of arterial disease. There is often a personal or family history of diabetes or arterial disease. Arterial disease often manifests at an earlier age in males and in individuals who smoke or have other risk factors.
- **B**lood pressure: Find out if it is elevated and if the patient is on medications.
- **C**holesterol: Elevated cholesterol is a risk factor, and the use of statin cholesterol-lowering agents may reduce this risk.
- **D**iet and obesity: Increased weight, especially a body mass index above 25, indicates an increased risk for heart and peripheral vascular disease as well as diabetes.

- **E**xercise: Individuals who exercise regularly have a lower risk of peripheral vascular disease and can build up a greater tolerance to overcome compromised circulation. In general, individuals with leg pain at rest or when recumbent in bed have rest pain that equates to severe ischemia, those who have pain or claudication (aching and throbbing calf muscles) with walking up a few stairs or less than 50 yards have severe disease, and individuals with symptoms after walking one or two blocks have moderate disease.
- **S**moking: One cigarette decreases circulation by 30% for 1 hour, and the more pack-years of accumulated smoking history, the greater the risk. Ask patients how many cigarettes they smoke a day and how many years they have been smoking (e.g., 30 years of smoking a half a pack a day is 15 pack-years [30 × 0.5 = 15 pack-years]).

Other risk factors include increased levels of homocysteine and hypothyroidism.[15] If peripheral vascular disease is present, it is also more common to have a history of coronary artery disease and previous stroke.[15]

Physical Examination

Skin inspection, palpation, and auscultation are all an important part of the physical examination. It includes examining the distal extremities for taut or shiny, atrophic skin that's present with arterial disease. Because skin color may indicate arterial perfusion, each toe should be noted and compared with the other foot and toes. Arterial insufficiency causes ischemic tissue to first become pale, progressing to a mottled netlike appearance (livedo reticularis) and subsequently to a dark purple hue and finally black (Fig. 17-5A and B). Elevating the foot at a 45-degree angle causes the ischemic limb to become pale. Immediately after positioning the ischemic foot in a dependent position, it becomes dark red or ruddy (dependent rubor or redness). This finding is the reactive hyperemia of ischemic tissue. There may be a loss of hair distally, and the nails may lose their luster and become thickened. Make sure to distinguish nail changes from changes that occur with a fungal infection (about half of all abnormal nails), psoriasis, or other nail disorders.

A

Figure 17-5. Arterial ulcer. **(A)** This photograph shows a necrotic great toe with blisters on the toes and foot, representing arterial insufficiency. **(B)** Mummified great toes. (B, Courtesy of J. Niezgoda.)

Palpate the skin for temperature changes. The skin of the distal part of an ischemic limb feels cool or cold, with temperature demarcation that correlates to the diseased artery. Infrared thermometers are now available for under $100 and should be utilized by every wound care clinician.[17] These less costly thermometers have been validated to the gold standard.[17] The thermometer will also identify repetitive trauma/deep inflammation in the neuropathic foot along with deep and surrounding infection.[18]

Capillary refill time is determined by compressing the skin (dorsum of the foot or toe pad) with the thumb to remove the local profusion

leading to a local blanching of color. Then, release the thumb to observe the capillary refill, and return of color may be a good indicator of arterial skin perfusion.[19,20]

The Wound Healing Society has cautioned us in their 2014 update on guidelines for arterial ulcers[21] that "the reliability of capillary refill time as an indicator of tissue perfusion is questionable." The guideline cautions that it is not a validated test. Capillary refill may still be a useful clinical test but should be used in conjunction with other clinical findings, and transcutaneous oxygen may be a good indicator of lower limb critical ischemia as it measures the sum total of the large vessel and local capillary circulation. Perform the capillary filling test with the foot slightly elevated. Normal capillary refill time is less than 2 seconds from pallor to normal skin color.[19]

Palpate pulses for presence, rate, regularity, strength, and equality. The most common objective physical finding is the presence or absence of pulses. Care must be taken when palpating pedal pulses. *It's common to mistake a contracting tendon for the presence of a pulse*. No universal consensus exists regarding a pulse grading system. According to the Inter-Society Consensus for the Management of Peripheral Arterial Disease (TASC II),[22] pulses are graded as 0 (absent), 1 (diminished), or 2 (normal). The American Heart Association's guideline adds a grade of 3 for bounding pulses. To ensure consistency, adhere to local policy. A high degree of inter-observer variability exists in determining the presence or absence of pulses. It can be confusing if clinicians report 2+ or 3+ pulse examinations. Communication is better facilitated if pulses are recorded simply as present or absent. However, even this seemingly obvious assessment parameter may not always be accurate. One study found only a 50% chance that two observers would agree with a third observer about the presence or absence of DP or PT pulses.[18] This same study identified the DP pulse congenitally absent in 4% to 12% of subjects and the PT pulse absent in 0.24% to 12.8%.[22] Additional descriptors of pulse, such as "weak" or "bounding," can be added to clarify your findings.

Practice Point

The best way to document pulses is to use descriptor terms, such as present or absent, rather than numerical ratings, such as 2+ or 3+. Use modifier words, such as weak or bounding, to further describe and clarify the pulse findings.

Although pulses in the foot may be present at rest, they may disappear with exercise. A patient who presents with claudication but has clearly discernable pulses should have an exercise test performed in the vascular laboratory. Clinicians are often tempted to skip the assessment of the elusive popliteal pulse, particularly when the DP and PT pulses are strong. While good pedal pulses indicate foot perfusion, finding bounding popliteal pulses may indicate a popliteal aneurysm. Popliteal aneurysms can be a source of emboli to the lower leg with resulting tissue or limb loss. These emboli are often the source of “blue toe syndrome” (Fig. 17-6A and B).

A

Figure 17-6. Blue toe syndrome. **(A)** Note “blue toe syndrome” in the second toe caused by tissue ischemia from arteriosclerosis. **(B)**“Blue toe syndrome” with forefoot tissue ischemia. (Photo courtesy of J. Niezgoda and M. Sieggreen.)

Patients with foot or leg ulcers should be tested for neuropathy. This is a common finding in persons with diabetes, but there are several other causes associated with a loss of protective sensation. Approximately two-thirds of patients with neuropathy have diabetes, but the other 1/3 of neuropathies are from other etiologies. For example, neuropathy commonly obscures a traumatic or pressure-induced wound in an ischemic limb. Lack of protective pain sensation and injury awareness prevents the patient with diabetes from seeking appropriate care early.

Evaluate neuropathy by testing light touch with monofilaments for the sensory component, examining for dry skin as part of the autonomic component, and eliciting reflexes for the motor component. You can remember to assess for neuropathy with the mnemonic SAM (**S**ensory, **A**utonomic, and **M**otor). An objective assessment of significant neuropathy is best performed by using the 5.07 Semmes-Weinstein monofilament.[23] To perform this assessment, ask the patient to close the eyes and indicate when he or she feels the monofilament. Test the areas over the plantar aspect of the first, third, and fifth toe; the first, third, and fifth metatarsal head; both sides of the plantar aspect of the midfoot; the plantar heel; and, lastly, the dorsum of the foot. Place the monofilament on the test position once until it bends slightly in a C-shape configuration, and then move it to the next position. Record the number of negative sites the patient reports; if there are four or more negative sites, then neuropathy is present, which indicates a loss of protective sensation (see Chapter 18 Diabetic Foot Ulcers and Fig. 18-3).

Practice Point

Use the mnemonic **SAM** to assess for neuropathy:

Sensory

Autonomic

Motor

Arterial Signs and Symptoms

Arterial insufficiency is commonly associated with complaints of pain[23] resulting from atherosclerotic arterial changes interrupting blood flow to tissues.[24–26] Claudication pain is pain occurring with exercise, which is relieved by rest, and is located in the muscle group distal to the stenosed or occluded artery. While the calf is the most common location for claudication, it can also occur in the buttocks, thighs, or feet and is predictable and reproducible. Claudication is described by patients as muscle cramping, aching, or weakness. The distance the patient is able to walk until the claudication first develops is referred to as the *initial claudication distance*. The distance the patient is able to walk before he or she has to stop is called the *absolute claudication distance* (Fig. 17-7). The period of time the patient needs for the pain to subside after he or she is forced to stop is referred to as the *recovery time*.

Figure 17-7. Absolute claudication distance. (Photo courtesy of A. Siddique.)

Practice Point

When taking a history, it's important to find out exactly how far the patient can walk before he or she needs to stop; a shorter distance indicates more severe atherosclerosis. Reported changes in ambulatory distance may indicate progressive atherosclerotic disease.

The patient with leg ulcers and poorly perfused tissue commonly seeks care because of sharp, severe, and possibly constant pain at the ulcer site and the distal extremity. Pain that occurs at rest represents inadequate perfusion and is a sign of threatened limb viability or critical limb ischemia. This pain is referred to as *rest pain*. The patient may describe waking at night with pain across the distal metatarsal area of the foot. In an attempt to relieve the pain, the patient will get out of bed and lower the foot to the ground (Fig. 17-8). This maneuver uses gravity to increase blood flow and improve tissue perfusion. The patient may even ambulate. The ischemic pain may be relieved by the small contribution of blood flow from collateral vessels if the limb is placed in a dependent position. The patient with pain at rest may begin sleeping with the legs in the dependent position that will predispose the distal leg to edema accumulation. Elevation of this edematous limb will further exacerbate rest pain, distinguishing it from venous insufficiency and other causes of edema.

Figure 17-8. Patient hanging foot off bed to relieve pain. (Photo courtesy of S. Baranoski.)

Practice Point

> Rest pain represents end-stage arterial insufficiency and usually requires revascularization.[16,25]

Patients with extensive sensory neuropathy—for example, those with diabetes—may not experience pain even with severely ischemic ulcers. On the other hand, these patients may experience such intense hyperesthesia associated with the neuropathy that they cannot bear the light touch of stockings. Ulcers in patients with neuropathy are typically found on the plantar side of the foot and are surrounded by calluses from long-term local pressure. These patients may describe the sensations of burning, stinging, shooting, and stabbing pain (neuropathic pain) rather than the more characteristic gnawing, aching, throbbing, and tender pain (nociceptive pain) associated with an acute injury of peripheral vascular disease.

When obtaining a history, note previous arterial surgery for vascular disease, including coronary artery disease and cerebrovascular disease. Vascular disease isn't limited to any one organ but can occur in all body systems: 60% of patients with peripheral arterial disease will have coronary artery disease, and 40% will have cerebrovascular disease. Document all medications, especially vasoconstrictor drugs. Ischemic symptoms are exacerbated by nicotine. Patients with symptomatic vascular disease may aggravate their symptoms by using tobacco, nicotine gum, or nicotine patches for smoking cessation.

Another arterial finding upon examination is gangrene. In ischemic tissue, gangrene initially appears pale, then blue-gray, followed by purple, and, finally, black. Gangrenous tissue eventually becomes black, hard, and mummified. The hardened tissue isn't painful, but significant pain may be present at the line of demarcation between the gangrene and the live but ischemic tissue. Gangrene may be a small skin lesion or extend to an entire limb depending on the location of the arterial lesion. If a small patch of skin is affected, the skin will dry and fall off, producing a skin ulcer or a healed tissue deficit. Large areas of gangrene may require debridement, skin graft, or potential amputation. *Do not attempt debridement prior to revascularization.*[22]

Additional physical findings are local patches of color change that may appear as small black or dark purple dots, circular areas found on

the distal toes, or localized infarcts around the toe nail beds (See Fig. 17-6A). Tissue changes found in these areas are caused by tissue ischemia from arteriosclerosis or by atheromatous debris embolizing from a proximal artery. Arterial erosions (loss of epidermis with an epidermal base) or deeper ulcers (loss of epidermis with a dermal or deeper base) may also be found between the toes, starting as a small, moist, macerated spot on the skin surface extending deep into the bony structure of the foot. This may also be caused by pressure due to ill-fitting footwear.

Arterial ulcers typically have distinct borders with a pale-gray or yellow-dry base. They may contain exposed tendons, fascia, fat, muscle, bone, or joint structures in their base. In contrast, venous ulcers most often have granulation tissue in their base and seldom demonstrate other structures found in the base of an arterial ulcer. The surrounding tissue may appear pale compared with skin elsewhere on the body, or it may be reddened if the leg is dependent. Chronic ischemic skin may appear thin and shiny. Foot elevation will produce skin pallor. The red or ruddy color of a dependent ischemic limb is called *dependent rubor* or *reactive hyperemia* (Fig. 17-9A and B). Even in a person of color, the difference in hue is discernible when the ischemic limb is compared with the contralateral well-perfused limb.

A

Figure 17-9. Dependent rubor. Foot elevation produces skin pallor in patients with ischemic skin (Buerger's sign or test). When dependent, the ischemic limb will have a red or ruddy color, as shown here in the patient's right leg. This is called dependent rubor **(A)** or reactive hyperemia **(B)** (Goldflam's sign). (Photos courtesy of J. Niezzgoda and M. Sieggreen.)

Arterial pressure is one of the most reliable physical findings in peripheral arterial disease.[25,26] However, lower-extremity blood pressures aren't obtained as a part of the routine physical examination. Bilateral brachial pressures should always be obtained on the initial examination to identify whether a discrepancy exists between them. The correct pressure is always the higher of the two pressures. This

pressure is used to determine the ankle–brachial pressure index (ABPI) when assessing lower-extremity perfusion.

Vascular Testing

Although an experienced vascular clinician can make a vascular diagnosis based on history and physical examination alone, vascular laboratory studies help pinpoint the diagnosis. The presence, location, and severity of arterial disease are confirmed by vascular laboratory procedures. Information obtained by vascular studies can predict potential ulcer healing (healable ulcer) when the cause is arterial insufficiency.[7] Laboratory tests differentiate among conditions contributing to a nonhealing ulcer.

Noninvasive vascular testing is divided into direct tests that image the vessel itself and indirect tests that demonstrate changes distal to the diseased vessel. These tests include segmental arterial Doppler ultrasound with pressures, arteriogram, ABPI, transcutaneous pressure of oxygen ($TcPO_2$), and toe pressures.[27]

Handheld Doppler Ultrasound

A Doppler ultrasound transmitting probe sends a signal that is reflected from an object to the receiving probe. If the signal strikes a moving object such as blood cells, a frequency shift is detected and reflected as sound (Doppler principle). The audible signals of arterial flow patterns can then be determined. The handheld Doppler is used to detect an audible signal on the dorsum of the foot or ankle (DP artery and PT artery). A blood pressure cuff is then placed around the lower calf and inflated until the audible signal disappears. The cuff is then slowly deflated, and when the signal returns, the systolic pressure is determined from the reading on the cuff gauge.

The ABPI may be falsely high in up to 80% of persons with diabetes and 20% of older individuals due to calcification of the lower leg arteries preventing constriction of the artery with inflation of the blood pressure cuff. Alavi and colleagues[28] measured the accuracy audible handheld Doppler (AHHD) ultrasound in 200 patients with 379 evaluable legs. The DP or PT artery was identified with the 8-MHz handheld Doppler, and the signs were categorized as monophasic or multiphasic (biphasic or triphasic). Multiphasic signals were related to ABPI segmental Doppler signals and toe pressures in a certified vascular lab as tabulated in Figure 17-10.

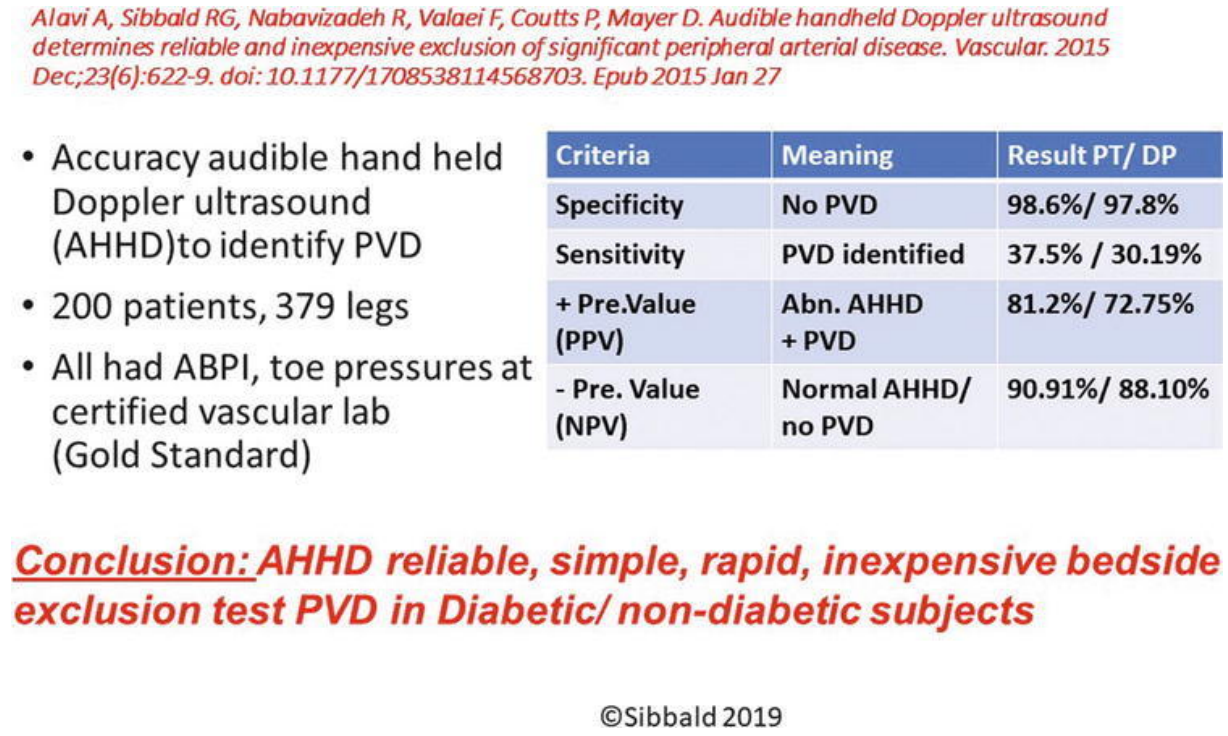

Figure 17-10. Audible handheld Doppler ultrasound determines reliable and inexpensive exclusion of significant PVD. (Photo courtesy of R. G. Sibbald, MD.)

The audible Doppler test is specific to rule out peripheral arterial disease of the lower legs, but it is not as sensitive as the duplex Doppler in the vascular lab to identify the level of the disease in the leg. There was also a high positive and negative predictive value for the test. The result is reported for a multiphasic wave is “the AHHD was greater or equal to 0.9.” This allows screening for the use of compression therapy for venous disease on the lower leg or to rule out large vessel arterial disease in most patients. Monophasic waves require referral to a certified vascular lab. The audible sounds are not influenced by arterial vessel calcification and AHHD reliable, simple, rapid, inexpensive bedside exclusion test to rule out PVD in diabetic/nondiabetic subjects. The study also calculated interrater reliability between a physician and a nurse assessing 20 of the patients independently. The interrater reliability between the two clinicians was 87.5% for the AHHD readings with a Cohen’s kappa of 0.76.

A second study by Tehan and Chuter[29] had 2 public and 2 private podiatrists evaluate 30 to 32 patients in the Australian public and private clinics. The study measured reliability using the Landis and Koch method with k > 0.75 = excellent agreement, > 0.40 to 0.75 = good agreement, and k < 0.40 = poor agreement. The study demonstrated low reliability (one value out of eight was in the good range) for the AHHD on patients (k 0.20 to 0.42) for inter- and intrarater reliability for the DP and PT artery. Audio recording of Doppler sounds gave poor to good interrater reliability (0.31 to 0.61) and poor to good agreement for intrarater reliability (0.53 to 1.00). Tracings of Doppler exam showing monophasic or multiphasic waves gave excellent interrater reliability (k 0.77 to 0.90) and intrarater reliability (k 0.77 to 0.81). The tested podiatrists have an experience of 1 to 8 years. They require more skills with a competency-based training program for the AHHD to match the interrater reliability from an expert clinic in the first cited study. Alternatively, the clinicians should purchase the more expensive 8-MHz handheld Doppler with the ability to record or show the waveforms on the bedside screen (Table 17-1).

Table 17-1

Criteria	Meaning	Result PT/DP
Specificity	No PVD	98.6%/97.8%
Sensitivity	PVD identified	37.5%/30.19%
+Pre.Value (PPV)	Abn. AHHD + PVD	81.2%/72.75%
–Pre.Value (NPV)	Normal AHHD/no PVD	90.91%/88.10%

Adapted from Alavi, A., et al. Audible Handheld Doppler Ultrasound Determines Reliable and Inexpensive Exclusion of Significant Peripheral Arterial Disease. *Vascular* 23(6):622-9, 2015. doi: 10.1177/1708538114568703. © Sibbald 2019.

Arteriogram

An arteriogram is an invasive test used to identify an operative lesion in the arterial system by outlining the patent arterial lumen (Fig. 17-11). Indications for a surgical procedure include incapacitating claudication, rest pain, nonhealing ulcers, and gangrene. An arteriogram is not indicated unless a bypass or dilation procedure is required. Some may not agree with this statement, given that CO_2 angiography and or intravascular ultrasound (IVUS) angiography may be required, if the patient cannot get noninvasive testing such as CTA or MRA. It is also not indicated when the patient is too ill for a surgical procedure or is refusing surgical intervention.[30]

Figure 17-11. Arteriogram. The arteriogram below shows iliac stenosis.

Arterial Testing

Propagation of a pulse wave originating in the heart is easily measured by auscultation of a peripheral artery with Doppler ultrasound. Recording the Doppler shift demonstrates the normal triphasic signal representing the three phases of the pulsation in a normal peripheral artery. The first wave represents forward flow of blood and arterial distention. The second phase represents the arterial relaxation and subsequent retrograde flow of blood. The third phase or portion of the triphasic Doppler signal is believed to represent the bulging of the aortic valve, which occurs during diastole. Some authors suspect that the third phase represents the rebound of the compliant, elastic arterial wall.

The third phase of the triphasic arterial signal is first lost as an artery becomes less compliant and is followed by loss of the second phase of the triphasic Doppler signal. With worsening occlusive disease proximal to the area of auscultation, the normally sharp first wave becomes flattened and broader. In severely diseased arteries, the Doppler signal can be a monophasic, low-amplitude wave. The minimum systolic pressure that can result in forward Doppler flow is used in the calculation of the ABPI, a measurement of arterial perfusion in the leg (Fig. 17-12).

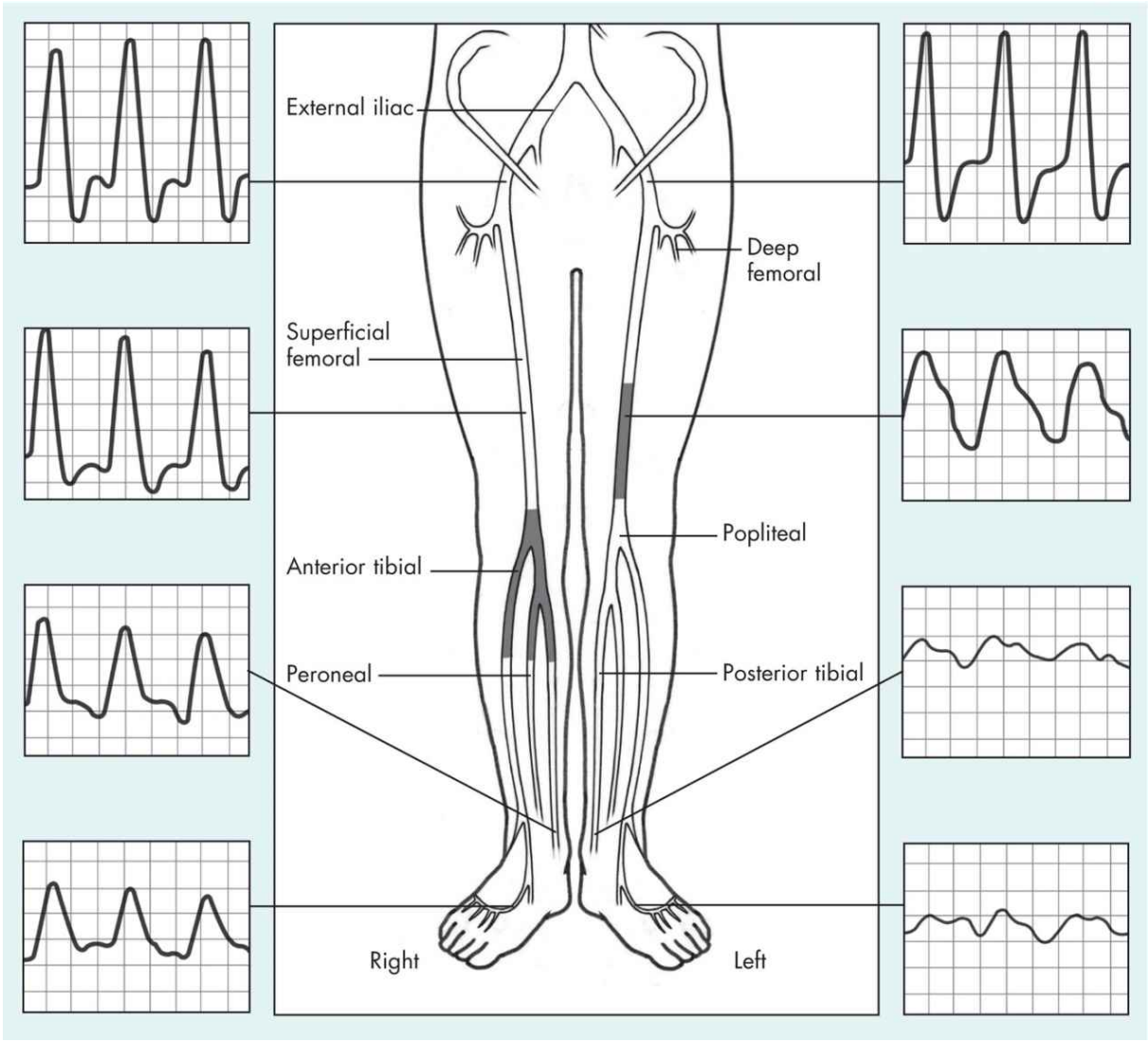

Figure 17-12. Arterial waveform changes. The arterial changes corresponding to occluded arteries are illustrated here.

Ankle–Brachial Index

Additional tests for arterial disease include ABPI, segmental pressures and waveforms, duplex ultrasound, and exercise treadmill for claudication. Perfusion is indirectly measured by the ABPI—the Doppler systolic pressure of the ankle artery divided by the brachial systolic pressure. ABPI ratios reflect the degree of perfusion loss in the lower extremity. If the higher leg pressure is 80 mm Hg and the higher arm pressure is 100 mm Hg, then the ankle–brachial pressure ratio (index) is 80/100 mm Hg or 0.80.

In most individuals, the resting ankle pressure in a supine position is equal to or greater than the brachial pressure, with an ABPI value of 0.9 to 1.3. An individual with claudication may have a normal ABPI in this position and have it drop during and after exercise. Patients with pain even in the resting state will have an abnormally low ABPI (<0.5). With exercise, the ABPI in the patient with rest pain usually doesn't fall because the arteries are already maximally dilated. Inadequate perfusion creates local tissue factors that result in vasodilation. Collateral pathways can't provide the additional tissue perfusion required, resulting in rest pain. A patient with ischemic tissue loss usually has a perfusion picture that is more consistent with rest pain than claudication[7] (Box 17-1).

Box 17-1 Obtaining an Ankle–Brachial Index

To obtain an ankle–brachial pressure index (ABPI), a sphygmomanometer and a Doppler device are needed. The procedure is performed as follows:

- Bilateral brachial Doppler pressures are obtained while the patient is supine. The higher of the two Doppler pressures is used as the brachial pressure in the ratio.
- The blood pressure cuff is placed on the leg just above the malleoli. The Doppler probe is placed at a 45-degree angle to the dorsalis pedis or the posterior tibial artery.
- The cuff is inflated until the Doppler signal is obliterated. With the Doppler probe over the artery, the cuff is slowly

deflated until the Doppler signal returns. The number is recorded as the ankle systolic pressure. The higher of the two systolic pressures in each leg is used as the ankle pressure for that leg.

- The higher of the ankle pressures is divided by the higher of the systolic brachial pressures. The ratio obtained is the ABPI.

(Photo courtesy M. Sieggreen.)

ABPI Interpretation

ABPI	Interpretation
0.9–1.3	Normal
0.75–0.90	Moderate disease
0.50–0.75	Severe disease
<0.5	Rest pain or gangrene
Unreliable	Diabetes

Segmental Pressures

Segmental pressures have been used since the 1950s to determine the location of arterial vascular lesions.[31] Pressures obtained at the level of the thigh, above the knee, calf, and ankle are compared with each other and with pressures in the other leg. An arterial lesion can be isolated with a 20-mm Hg gradient between cuff pressures. If no pressure gradient exists on a limb that claudicates, the patient is asked to exercise, and repeat pressures are obtained.

A palpable pulse indicates an arterial flow pressure of approximately 80 mm Hg or more in the foot. With calcification of the intima, the arterial pressures derived in the larger vessels of the leg can be falsely elevated. If an ABPI is over 1.3, the results are not reliable. The vascular laboratory will then have to rely on accessory tests, such as the toe pressure procedure or $TcPO_2$ test.

Toe Pressures

In patients with severe atherosclerosis (diabetes, chronic renal failure, advanced age), the tibial vessels become circumferentially calcified, which renders them incompressible. Toe pressure tests measure the flow through the large toe where the vessel is small enough that calcium deposits don't circle the entire vessel and compressibility is usually present. A toe pressure of 55 mm Hg or higher, even in a person with diabetes, is usually adequate for healing. Toe pressures of 30 to 55 mm Hg usually indicate some vascular compromise, and the wound healing will be more difficult. Pressures below 30 mm Hg may be adequate if the skin is intact, but as soon as injury results and disrupts the cutaneous barrier, the vascular supply is often inadequate for the repair process.[32]

Evidence-Based Practice

A falsely high pressure reading is commonly seen in patients with diabetes due to incompressible artery walls caused by medial sclerosis of the arteries.[10,32] When the vessels are incompressible, toe pressures are obtained because they're reported to be more accurate.

Treating Vascular Ulcers

When treating vascular ulcers, follow the “preparing the wound bed” paradigm below[32–34]:

- Treat the cause: bypass, stents, or dilation with a consult to a vascular specialist
- Patient-centered concerns: pain, quality of life, and activities of daily living
- Local wound care:
    - Healable wound: debridement, moisture balance, and bacterial balance
    - Maintenance wound: procedures may be more conservative because of patient or system factors preventing the wound from healing
    - Nonhealable wound: requires conservative debridement, moisture reduction, and bacterial reduction

With a healable wound, debridement of slough or nonviable tissue is actively promoted to create a clean wound. This may include careful sharp surgical debridement (with bleeding after debridement) or the use of mechanical, enzymatic, or autolytic debridement methods with dressings (usually alginates, hydrogels, or hydrocolloids).

A maintenance wound—one that could heal but patient factors such as smoking, inconsistent treatment, excessive obesity, or uncontrolled diabetes may make sustained healing less likely—requires conservative, superficial debridement (with no bleeding after debridement), accompanied by local wound care for bacterial and moisture balance. If a wound doesn’t have enough blood supply to heal, the surface of the wound or necrotic gangrenous tissue should be allowed to dry and demarcate. This can be facilitated by removing the soft slough around the proximal intersection of the necrotic and viable tissue but leaving the necrotic cap intact. Moisture and bacterial reduction can be reduced with acceptable tissue toxicity using antiseptic agents, including povidone–iodine or chlorhexidine.[32,35–38] A derivative of chlorhexidine, PHMB, polyhexamethylene biguanide, may be safer especially around the eye and ear where chlorhexidine is

contraindicated and PHMB is the most common preservative in eye and ear topical formulations. These agents have a broad spectrum of action, a sustained residual effect, and acceptable tissue toxicity for this indication. Agents such as sodium hypochlorite, quaternary ammonium agents, and various aniline dyes (mercurochrome) have higher cellular toxicities and more limited antibacterial effects.[32,35–38]

Wound Infection

It is important to remember that all chronic ulcers contain bacteria (contamination). When bacteria are attached to tissue and multiply, they become colonized and can lead to damage that delays healing (may be called local infection, critical colonization, increased bacterial burden, covert infection, or superficial infection). Patients with critical colonization don't have all the classic signs and symptoms of a deep tissue infection. A bacterial swab does not diagnose infection but will identify the organisms present and their sensitivities to antimicrobial therapy. The bacterial swab will not identify biofilm associated organisms or their antimicrobial sensitivities.

The superficial compartment of the wound bed should be examined for more than one sign of bacterial damage. The key features of a wound bed can be remembered by the NERDS mnemonic[33,35] (see NERDS© and STONEES© in Chapter 7, Wound Bioburden and Infection).

Topical treatment for healable wounds that are critically colonized wounds could include the various new silver dressings, cadexomer iodine, or two nonantiseptic releasing foams—polyhexamethylene biguanide (PHMP) and methylene blue/crystal violet. Povidone–iodine or chlorhexidine may be considered for wounds with inadequate blood supply to support healing.

Deep tissue infection requires systemic antimicrobial agents. The classic signs of warmth, tenderness, swelling, and erythema can be supplemented for persons with chronic wounds by the mnemonic STONEES[33,35] (see NERDS© and STONEES© in Chapter 7, Wound Bioburden and Infection). If exudate and odor are present, other criteria are needed to determine whether the infection is superficial or in the deep and surrounding tissue.

Practice Point

- The diagnosis of superficial or deep and surrounding tissue infection can be made clinically with a bacterial swab to help with treatment decisions.
- Persons with diabetes, neuropathy, and foot ulcers often have a false-negative x-ray of the foot for osteomyelitis, and other clinical criteria, including probing or exposed bone, should be used to suspect osteomyelitis.
- The use of a bone scan is limited because it is expensive and may give false-positive results in the presence of inflammation. Magnetic resonance imaging (MRI) may be more helpful diagnostically.

Wound Cleansing

A clean wound, free from dead tissue and wound debris, is necessary for healing to occur. Many commercial wound cleaners have some cytotoxicity, but they have surfactant properties that are often useful. Povidone–iodine, chlorhexidine, hydrogen peroxide, and 0.25% acetic acid have been shown to interfere with fibroblast formation and epithelial growth.[32,33] The selective use of these agents, particularly povidone–iodine and chlorhexidine, should be reserved for wounds that don't have the ability to heal or for time-limited use in wounds in which bacterial burden is more important than cellular toxicity.[32,36,37]

Evidence-Based Practice

The advantage of wound cleansing should be weighed against damaging new tissue growth.[38]

The safest wound cleanser is 0.9% saline solution or water. Wounds should be cleaned with a force strong enough to dislodge debris but gentle enough to prevent damage to newly growing tissue. The pressure to accomplish this goal ranges from 4 to 15 pounds per square inch (psi).[39] A 19-gauge needle or 19-gauge angiocatheter distributes approximately 8 psi when used with a 35-mL syringe. A commercial cap on a saline irrigation bottle is a less expensive method to distribute adequate pressure. Hydrotherapy or whirlpool debridement has been used to aid in cleaning and debridement of arterial ulcers. However, the risk of infection outweighs the benefits of whirlpool for debridement and is not currently recommended.[38–40] This may suggest that vigorous irrigation may be a significant factor in cleansing the wound, but the potential harm must be considered along with the benefits. (For more information about wound cleansing, see Chapter 7, Wound Bioburden and Infection.)

Dressings chosen for specific wounds depend on the wound bed condition and the goal for the wound. Many new dressings are designed to support moist wound healing (see Chapter 9, Wound Care & Treatment Options). Because skin is fragile in patients with either arterial or venous disease and can be easily injured, tape and adhesive products should be used with extreme caution. The use of methods to secure dressings that minimize skin injury is recommended. Of the available adhesive products, soft silicones are less likely to cause local trauma during dressing changes. Products without adhesives that secure dressings are the first choice when they are available.

Practice Point

The preferred attachment device for dressings on vascular leg ulcers is a gauze roll or commercial devices (such as netting or tube gauze) that hold dressings in place without adhesive that can damage fragile skin.

Arterial Ulcer Treatment

Treatment of arterial ulcers includes increasing the blood supply to the area. Positioning the extremity in a dependent position may facilitate blood flow by gravity through collateral vessels. Use caution if devices such as a foot cradle are used for protection because an insensate foot is subject to trauma from the cradle's hardwood or metal (Fig. 17-13). Debridement of nonviable tissue should not be performed in the presence of ischemia because the blood flow is insufficient to heal the new surgical wound. Ulcers without adequate arterial inflow must be kept dry—in contrast to the principle of moist wound healing for ulcers with adequate blood supply (Fig. 17-14). Moisture provides a bed for bacterial growth if eschar, slough, or gangrenous tissue is present. This tissue, if kept dry, can be left in place until demarcation or debridement is indicated.

Figure 17-13. Patients with ischemic tissue who are insensate often don't seek help until extensive damage has occurred. (Photo courtesy of M. Sieggreen.)

Figure 17-14. Keep dry: arterial ulcer on forefoot. (Photo courtesy of M. Sieggreen.)

Ulcers with adequate blood supply that are expected to heal should be dressed with products that support moist wound healing principles.[32] These dressings include hydrocolloids, thin films, foams, and, if nothing else is available, moist saline gauze. The surrounding intact tissue should be protected from fluid accumulation, which can macerate the healthy skin at the ulcer border. Consider using a barrier

such as film forming liquid acrylates, petrolatum, zinc oxide, or windowed dressings.

Arterial reconstruction is the treatment of choice to improve the circulation for most patients.[24] Treatment for arterial leg ulcers requires re-establishing arterial inflow before any other treatment is established. This is usually preceded by a noninvasive vascular test and an arteriogram (computerized tomography angiogram, magnetic resonance angiogram, digital subtraction angiogram) followed by angioplasty and/or surgery. Simultaneously, local ulcer treatment can be determined. Usually, the arterial ulcer has a dry ulcer bed (see Fig. 17-14). The patient may have several punctate ulcers with regular borders as well as dry eschar or gangrene distal to the most perfused tissue—usually the tips of the toes or an entire toe. This tissue must be kept dry until adequate arterial perfusion occurs. Moistened gangrenous tissue can provide a medium for bacterial growth (Fig. 17-15).

Figure 17-15. Keeping gangrene dry. Gangrenous tissue must be kept dry until adequate arterial perfusion is restored to the area. In the photograph above, the necrotic toes are left open to the air with alcohol wipes placed between them to promote drying.

Surgical Treatment for Arterial Ulcers

Surgical treatment should be considered when patients have incapacitating claudication, rest pain, nonhealing ulcers, or progressive gangrene and infection that cannot be controlled. For arterial ulcers, surgical treatment is aimed at restoring tissue perfusion. Bypass grafting may be performed using autologous veins or, when autologous veins are not available, prosthetic grafts, either reversed or in situ. Despite the fact that endovascular techniques are not superior to surgical techniques with regard to vessel patency, wound healing and limb salvage can be attained by using endovascular techniques for patients previously considered ineligible for revascularization. There used to be generally less sustainable long-term results from percutaneous balloon angioplasty and stent insertions, atherectomy (percutaneous endoluminal removal of atherosclerotic plaque),[41] and laser ablation of atherosclerotic lesions,[42] except in the common iliac arteries. Since the use of drug-eluting stents (DES), studies have shown restenosis rates have decreased and 5-year patency rates were sustained, including complex lesions in the SFA.[43–45] These minimally invasive procedures may be very useful in the high-risk patient, and they expand treatment options.

Large arterial ulcers may require skin grafting to close the defect, but this can only be successful if combined with revascularization procedures. The BASIL (Bypass Versus Angioplasty in Severe Ischemia of the Leg) trial,[43] which compared bypass surgery and angioplasty, clearly showed that bypass surgery was superior in achieving amputation-free survival. Also, those patients who underwent bypass surgery first fared better than those who underwent angioplasty first. However, this superiority was not significant until after 2 years. The BASIL trial[43,46] also showed that autologous veins were superior to prosthetic conduits for these bypasses. Unfortunately, the trial did not include the much more common practice of hybrid procedures, combined bypass surgery, and endovascular intervention. Nevertheless, it reinforces the long-held concept in limb salvage surgery that being aggressive is usually better for the patient.

The treatment of ulceration due to arterial insufficiency in the lower extremities depends on the level that the occlusive disease occurs. Surgical procedures for arterial insufficiency are generally grouped into three major areas:

- Aortoiliac bypass
- Femoropopliteal bypass
- Distal bypass

Restoring Tissue Perfusion

Occlusive disease in many patients is multileveled. The rule of thumb is to improve inflow first in these patients and then, if necessary, perform an outflow procedure. Inflow usually involves the aortoiliac segments. The exact surgical operation is tailored to the individual patient's physiologic status and need. For example, an elderly, frail patient with severe aortoiliac occlusive disease may not be a candidate for an aortobifemoral bypass graft. In this type of patient, an extra-anatomic axillobifemoral bypass graft is considered. By avoiding intra-abdominal surgery and clamping of the abdominal aorta, the overall morbidity for these surgeries can be reduced. However, the trade-off for this is that an axillobifemoral bypass graft generally doesn't have the long-term patency rates than an aortobifemoral bypass graft does.

Percutaneous Balloon Angioplasty

The development of percutaneous balloon angioplasty, with or without stent placement, has significantly reduced the need for routine aortobifemoral bypass surgery in patients with aortoiliac occlusive disease.[47] Isolated short-segment stenoses can be treated successfully with balloon angioplasty. Short-segment stenoses are generally defined as those less than 10 cm in length, commonly less than 5 cm. With advances in stent development, acute occlusions occurring as a result of atherosclerotic plaque rebound have decreased. The long-term patency rate for stents approaches that for arterial bypass but only in the aortoiliac segments.[48] Good long-term outcomes with bare metal stents are still haunted by the occurrence of in-stent restenosis. The development of covered stents, with graft material (usually polytetrafluoroethylene [PTFE]) either on one side of the stent metal or enclosing it completely, may hold the answer.[49] Infrainguinal balloon angioplasty with or without stent placement is still inferior to surgical intervention. However, the results continue to improve after development of drug-eluting SFA stents, as referenced earlier.[43–45] This procedure still holds a place in the treatment of high-risk patients.

According to the TASC II Guidelines,[22] when possible, arterial reconstruction by means of endovascular techniques should be considered before more invasive surgical techniques. Percutaneous intraluminal balloon angioplasty and stenting are considerations for arterial stenoses and occlusions classified as TASC A or B lesions. TASC C and D lesions are usually longer and more extensive and often require bypass operations (again, TASC C and D lesions appear to be treated more commonly with endovascular techniques). Newer hybrid procedures include a combination of open surgical and endovascular techniques.[22]

Femoropopliteal Bypass Graft

A femoral–popliteal bypass graft is the standard treatment for femoropopliteal disease. In contrast to an aortoiliac bypass, where the bypass conduit is that of a synthetic material, the femoropopliteal segment may have either a prosthetic conduit or an autogenous venous conduit. The patency rate for a bypass of the femoral popliteal segment depends upon the choice of conduit and the distal level of the bypass. In an above-knee femoropopliteal surgery, the patency rate between autogenous vein and prosthetic material has no significant difference, although long-term patency rates are better when an autogenous venous conduit is used. In a below-knee femoropopliteal bypass, prosthetic material is far inferior to that of autogenous venous conduits.[50] An autogenous venous conduit should be used in the below-knee position whenever possible (Table 17-2).

Table 17-2 Graft Patency Rates

Type of Graft	1 Year	2 Years	3 Years	4 Years
Above-Knee Femoropopliteal Grafts				
Reverse saphenous vein	84%	82%	73%	69%
Polytetrafluoroethylene (PTFE)	79%	74%	66%	60%
Below-Knee Femoropopliteal Grafts				
Reverse saphenous vein	84%	79%	78%	77%
PTFE	68%	61%	44%	40%
Limb salvage				
Reverse saphenous vein	90%	88%	86%	75%
Reverse saphenous vein	94%	84%	83%	
Infrapopliteal Grafts				
Reverse saphenous vein	84%	80%	78%	76%
PTFE	46%	32%	21%	82%
Limb salvage				
Reverse saphenous vein	85%	83%	82%	48%
PTFE	68%	60%	56%	
At or Below-Ankle Grafts				
Reverse bypass vein	85%	81%	76%	
In situ vein bypass	92%	82%	72%	
Foot salvage	93%	87%	84%	

This chart shows the percentage of grafts that remain patent after 1, 2, 3, and 4 years.

Below-knee femoropopliteal bypasses using veins have a higher patency rate than above-knee femoropopliteal bypasses because a certain amount of atherosclerotic disease at the level of the knee joint can be missed if only anteroposterior arteriography views are obtained. For this reason, many vascular surgeons require oblique views of the popliteal artery so as to preclude this as a source of decreased long-term patency rates.

Distal Bypass

A distal bypass, below the tibial peroneal trunk, requires an autogenous venous conduit. It's reserved for patients with tissue loss when pulsatile arterial perfusion to an ischemic area is desired. Although somewhat controversial, either a reversed venous bypass or an in situ bypass can be performed. Patency rates were equivalent in large series comparing these two techniques.[50] The in situ technique is generally reserved for patients with considerable size disparity between the proximal and distal venous conduit, such as the greater saphenous vein. An in situ bypass is technically more demanding and requires more operative time than a reversed venous bypass. Some vascular surgeons advocate the creation of a controlled arteriovenous fistula in order to promote long-term patency rates of the prosthetic conduit, when these are used for distal bypasses. However, autologous veins are considered superior to prosthetic vessels in terms of long-term patency.

A patient with calf claudication requires improved perfusion to the posterior calf muscles. Claudication can occur in the buttocks, thighs, or isolated compartments of the lower legs. The perfusion of the respective symptomatic musculature is what determines the level of the outflow portion of the bypass. In patients with combined aortoiliac superficial femoral popliteal disease, 90% of the claudication can be improved by merely improving the inflow to the profundal system by some form of aortoiliac bypass. It is for this reason that routine combined aortofemoral and femoropopliteal bypasses should be avoided. In patients with lifestyle-limiting claudication with isolated superficial femoral artery disease, a femoropopliteal bypass is usually all that is needed.

Patients with ischemic tissue loss usually require pulsatile arterial flow to heal their lesions. If these lesions are in the foot, then whatever bypass is necessary to restore pulsatile arterial flow to the affected area should be performed. Combined with the appropriate vascular bypass procedure, an area of ischemic tissue loss with gangrenous edges should be debrided to viable tissue. In some patients, however, if there is dry gangrene, autoamputation can be anticipated once

adequate perfusion is restored. Some clinicians allow the gangrenous eschar to autoamputate to enable normal epithelial coverage of the underlying eschar before eschar separation. If, however, the area of tissue loss involves a digit, amputation with primary closure may be recommended if no infection is present. This can be performed in conjunction with the vascular bypass procedure, or the procedures can be separated by several days if deemed appropriate.

Arterial reconstruction with an in situ graft may be used to revascularize the lower extremity well below the knee. An in situ graft is a vein left in its natural location, anastomosed to the arterial system above and below the arterial stenoses, after the valves are lysed. This procedure allows the surgeon to reconstruct the smaller distal arteries in the lower extremity near the foot. These reconstructed vessels are close to the skin surface. The surgeon must use extreme care not to cause injury to underlying vessels when using sharp debridement for the necrotic ulcers. Autolytic debridement is a safer debriding alternative.

Medical Treatment

Medical treatment of arterial disease may include antiplatelet drugs, such as aspirin or clopidogrel, which inhibit the binding of adenosine triphosphate (ATP).[51] Clopidogrel was shown to be slightly better than aspirin in a comparative study. In addition, cilostazol[52,53] has been used not only to decrease platelet aggregation but also to act as a vasodilator that may facilitate an increase in exercise capacity. However, it cannot be used in patients with heart failure. In addition, building up exercise tolerance with a conditioning program may also be important. The 2014 Wound Healing Society Guidelines[21] report that pentoxifylline does not improve arterial ulcer healing.

Measuring Healing

Calculating healing rates is problematic when no standard measurement for wound healing parameters exists. Following wounds to complete healing is one method, but this method is not satisfactory if changes in therapy are needed. Healing rates can be expressed as percent of ulcer area, measurement of change in ulcer perimeter, or percent of ulcer area healed. However, the perimeter and surface area are much greater in large ulcers. Using these measurements will give erroneously high healing rates for larger ulcers compared with smaller ulcers. For example, if the percent of ulcer healed is used as a measurement, smaller ulcers will appear to heal faster than large ulcers by comparison.

Another method traces ulcers on a celluloid screen and then measures them over time. The area and circumference of the tracing are calculated by a computer program. In general, a healing trajectory will be established if a wound is 20% to 40% smaller by week 4, and that wound should heal by week 12 provided that the same healing rate is maintained.[54]

There are some patients whose ulcers do not heal at the expected rate. If tissue damage has progressed beyond salvage, surgery is too risky, or if the limitations of the ischemic limb are interfering with quality of life, amputation may be considered.

Patient Education

The patient may inadvertently neglect the ulcer or fail to use preventive measures if the nature of the condition is not understood. Patient education includes the reason for the ulcer and the treatment rationale. Patient-centered concerns should be central to the treatment process, and active patient involvement includes the recognition and reporting of changes that indicate problems with healing. Patient and family education includes assessment of patient and family needs and level of comprehension about the arterial ulcer and its etiology. Teaching methods vary and are chosen specifically to facilitate the most appropriate method for each patient and family.

Risk Factors

Factors that increase risk for arteriosclerosis include smoking, diabetes (with elevated HbA1C), hyperlipemia, and hypertension.[53] Smoking is a risk factor in 73% to 90% of patients with atherosclerotic arterial disease.[53] Up to 30% of patients with arterial disease are reported to have diabetes,[55] and 16% to 58% of patients with diabetes have arterial disease.[56,57] Hypertension is present in 29% to 39% of patients with atherosclerosis, and 31% to 57% of patients with atherosclerosis have hyperlipidemia.[58] Risk factor modification is part of the treatment for vascular ulcers to reduce the possibility of further breakdown.

Patients can help themselves by positioning and reducing activities that impair blood flow. After a surgical or percutaneous intervention to restore arterial flow, the patient should continue behaviors that promote vascular health and reduce risk factors (see Patient Teaching: Teaching about arterial ulcers).

Patient Teaching*Teaching About Arterial Ulcers*

Teach the patient with an arterial ulcer to:

- monitor arterial or graft patency by palpating pulses
- recognize signs and symptoms of graft failure and what to report
- avoid nicotine in any form, including secondhand smoke
- begin or maintain a regular exercise program
- manage blood glucose, if diabetes is present
- control hyperlipidemia
- manage hypertension
- reduce weight, if indicated
- perform meticulous foot care
- manage ulcer care.

Smoking Cessation

Smoking cessation is critical for patients with arterial insufficiency. The direct relationship between tobacco use and ischemia is well known. Smokers are more likely to develop claudication than nonsmokers. However, the link between smoking and vascular disease isn't well recognized by many patients. In one study, only 37% of smokers with peripheral vascular disease understood the strong association between smoking and vascular disease.[53] Patients must be informed of the negative effects of smoking on the vascular system. They should be referred to smoking cessation specialists if needed. Teach the patient the ABCDES mnemonic to remember the risk factors of arterial disease.[10]

Summary

Success in managing arterial ulcers requires a total patient commitment. Risk factors and ulcer management are so dependent upon the patient's activities that the patient must have as much information as possible to actively participate in the treatment process. An understanding of the peripheral vascular blood supply and the need for adequate tissue oxygenation is critical to the management of arterial ulcers in the legs and feet.

Arterial reconstruction is the hallmark of treatment for arterial disease. In general, dry arterial ulcers or those with fixed, stable, dry eschar should be kept dry until the tissue is revascularized. Economic concerns make it imperative to choose the appropriate dressings and treatment. Research demonstrates little increased healing rate benefit of the newer topical wound treatments over the old gauze dressings. Some modern wound dressings provide other cost-effective benefits along with decreased nursing time, improved quality of life, and reduced pain issues.

PATIENT SCENARIO

Clinical Data

Mr. WG was an 82-year-old Caucasian male who presented with a foot ulcer (Fig. 17-16A). His past medical history was significant for insulin-dependent diabetes, hypertension, and chronic renal disease but not on dialysis, chronic obstructive airway disease, severe ischemic heart disease, and extensive peripheral arterial disease. He was a previous smoker but did not smoke at the time of the assessment.

A

B

C

D

E

Figure 17-16. (A) Arterial foot ulcer. **(B)** Photo of monoplace hyperbaric chamber. **(C–E)** Progression of surgical debridement. **(F)** Healed arterial foot ulcer. (Photos courtesy of M. Sieggreen.)

His surgical history included two previous coronary bypass procedures. It was determined that he would need a femoropopliteal bypass procedure and his lesions were not amenable to endovascular intervention. He was evaluated by an anesthesiologist and a cardiologist and was determined to be too high a risk for general anesthesia because of his cardiac status.

The option of hyperbaric oxygen therapy was considered, and he was in agreement with this plan (Fig. 17-16B). His $TcPO_2$ levels increased from resting pressures of 5 mm Hg (on room air) to 28 mm Hg after 15 minutes on a nonrebreather oxygen mask.

Debridement of his foot was done during his hyperbaric oxygen therapy (Fig. 17-16C–E). After 15 sessions of hyperbaric oxygen therapy, his $TcPO_2$ at rest increased to 23 and 45 mm Hg on a nonrebreather mask. He was able to return to his own home with intensive wound care performed by a wound care practitioner and home oxygen therapy.

On subsequent follow-up visits, he reported a remarkable improvement of his foot and quality of life (Fig. 17-16F). Unfortunately, he passed away, due to his underlying cardiac disease, within a year but avoided amputation and was able to function within his home.

Show What You Know

1. **Risk factors for the development of arterial ulcers include all of the following except:**
   A. smoking.
   B. hypercholesterolemia.
   C. diabetes mellitus.
   D. varicose veins.
   E. hypertension.

2. **Patients with arterial ulcers that do not have adequate blood supply to heal should have local wound care that includes:**
   A. aggressive local debridement to bleeding tissue.
   B. silver dressings that promote moisture balance.
   C. local antiseptics such as povidone–iodine and chlorhexidine.
   D. moisture balance dressings such as a hydrogel.

3. **Which of the following is most likely to be associated with an arterial ulcer?**
   A. Lipodermatosclerosis
   B. Reduced blood flow
   C. Edema
   D. Systemic hypertension
   E. Diabetes mellitus

4. **The ankle–brachial pressure index (ABPI) is an indicator of loss of perfusion in the lower extremity.**
   A. True
   B. False

5. **Surgical treatment for arterial ulcers most commonly includes:**
   A. a graft.
   B. valvoplasty.
   C. a bypass graft.

D. phlebectomy.

Answers to these questions are found on pages 685 and 686.

References

1. Young, J.R. “Differential Diagnosis of Leg Ulcers.” Cardiovascular Clinics 13(2):171-93, 1983.
2. Cornwall, J.V., et al. “Leg Ulcers: Epidemiology and Aetiology.” British Journal of Surgery 73(9):693, September 1986.
3. Coon, W.W., et al. “Venous Thromboembolism and Other Venous Disease in the Tecumseh Community Health Study.” Circulation 48(4):839-46, October 1973.
4. Dewolfe, V.G. “The Prevention and Management of Chronic Venous Insufficiency.” Practical Cardiology 6:197-202, 1980.
5. Callam, M.J., et al. “Chronic Ulcers of the Leg: Clinical History.” British Medical Journal (Clinical Research Edition) 294(6584):1389-91, May 1987.
6. Nelzen, O., et al. “The Prevalence of Chronic Lower-limb Ulceration Has Been Underestimated: Results of a Validated Population Questionnaire.” British Journal of Surgery 83(2):255-8, February 1996.
7. Rutherford, R.B. “Patient Clinical Evaluation.” In J.L. Cronenwett, K.W. Johnston, eds. Rutherford Vascular Surgery, Vol. 1, 8th ed. Philadelphia, PA: Elsevier Saunders, 2014.
8. Moore, W.S., ed. Vascular and Endovascular Surgery: A Comprehensive Review, 8th ed. Philadelphia, PA: WB Saunders, Elsevier, 2013.
9. Bergan, J.J., Bunke-Paquette N., eds. The Vein Book. New York: Oxford University Press, 2014.
10. Weir, G.R., Smart, H., vanMarle, J., et al. “Arterial Disease Ulcers, Part 1: Clinical Diagnosis and Investigation.” Advances in Skin & Wound Care 27(9):421-8, 2014.
11. Forsgren, A., Nelzen, O. Changes in the Etiological Spectrum of Leg Ulcers after a Broad-scale Intervention in a Defined Geographical Population in Sweden. European Journal of Vascular and Endovascular Surgery 44:498-503, 2012.
12. Wood, A.J.J., Hiatt, W.R. “Medical Treatment of Peripheral Arterial Disease and Claudication.” New England Journal of Medicine 344(21):1608, May 2001.
13. Mustoe, T.A., O’Shaughnessy, K., Kloeters, O. “Chronic Wound Pathogenesis and Current Treatment Strategies: A Unifying Hypothesis.” Plastic and Reconstructive Surgery 117(7 Suppl):35S-41S, 2006.
14. Hiatt, W.R., Brass, E.P. “Pathophysiology of Peripheral Artery Disease, Intermittent Claudication, and Critical Limb Ischemia.” In M.A. Creager, J.A. Beckman, J. Loscalzo, eds. Vascular Medicine: A Companion to Braunwald’s Heart Disease, 2nd ed. Philadelphia, PA: Elsevier Saunders, 2013:223-30.
15. Sibbald, R.G., Ayello, E.A. “Assessing Arterial Disease History Using ABCDE’s Mnemonic.” Presented at School of Nursing, Wenzhou University Wound Care Symposium, Wenzhou, China, November 2009.

16. Aronow, W.S. “Management of Peripheral Arterial Disease.” Cardiology in Review 13(2):61-8, March-April 2005.
17. Mufti, A., Coutt, P., Sibbald, R.G. “Validation of Commercially Available Infrared Thermometers for Measuring Skin Surface Temperature Associated with Deep and Surrounding Wound Infection.” Advances in Skin & Wound Care 28(1):11-6, January 2015.
18. Sibbald, R.G., Mufti, A., Armstrong, D.G. “Infrared Skin Thermometry: An Underutilized Cost Effective Tool for Routine Wound Care Practice and Patient High Risk Diabetic Foot Self-monitoring.” Advances in Skin & Wound Care 28(1):37-44, January 2015.
19. Swartz, M.H. Chapter 12: The Peripheral Vascular System. In M.H. Swartz, ed. Textbook of Physical Diagnosis, 7th ed. Philadelphia, PA: Elsevier Saunders, 2014.
20. White, C.J. Chapter 79: Atherosclerotic Peripheral Arterial Disease. In L. Goldman, A.I. Schafer, eds. Goldman-Cecil Medicine, 25th ed. Philadelphia, PA: Elsevier Saunders, 2016.
21. Federman, D.G., Ladiiznski, B., Dardik, L. et al. Wound Healing Society 2014 Update on Guidelines for Arterial Ulcers: Guidelines for the Treatment of Diabetic Ulcers. Wound Repair and Regeneration 24(1):127-35, 2016. doi: https://doi.org/10.1111/wrr.12395.
22. Norgren, L., Hiatt, W.R., Dormandy, J.A., et al. “Inter-Society Consensus for the Management of Peripheral Arterial Disease (TASC II).” Journal of Vascular Surgery 45(Suppl S):S5-67, 2007.
23. Mayfield, J.A., Sugarman, J.R. “The Use of the Semmes-Weinstein Monofilament and Other Threshold Tests for Preventing Foot Ulceration and Amputation in Persons with Diabetes.” Journal of Family Practice 49(11 Suppl):S17-29, November 2000.
24. DePalma, R.G. “Atherosclerosis: Pathology, Pathogenesis, and Medical Management.” in W.S. Moore, ed. Vascular and Endovascular Surgery: A Comprehensive Review, 8th ed. Philadelphia, PA: Elsevier Saunders, 2013.
25. Fahey, V.A., White, S.A. “Physical Assessment of the Vascular System.” in V.A. Fahey, ed. Vascular Nursing, 4th ed. Philadelphia, PA: WB Saunders, 2003.
26. Beckman, J.A., Creager, M.A. “The History and Physical Examination.” In M.A. Creager, J.A. Beckman, J. Loscalzo, eds. Vascular Medicine, 2nd ed. Philadelphia, PA: Elsevier Saunders, 2013.
27. Ruangsetakit, C., Chinsakchae, K., Mahawongkajit, P., et al. “Transcutaneous Oxygen Tension: A Useful Predictory of Ulcer Healing in Critical Limb Ischemia.” Journal of Wound Care 19(5):202-6, May 2010.
28. Alavi, A., et al. “Audible Handheld Doppler Ultrasound Determines Reliable and Inexpensive Exclusion of Significant Peripheral Arterial Disease.” Vascular 23(6):622-9, 2015. doi: 10.1177/1708538114568703.
29. Tehan, P.E., Chuter, V.E. “Use of Hand-Held Doppler Ultrasound Examination by Podiatrists: A Reliability Study.” Journal of Foot and Ankle Research

8(1):36, 2015. doi: 10.1186/s13047-015-0097-2.
30. Brantigan, C.O. “Peripheral Vascular Disease: A Comparison between the Vascular Laboratory and the Arteriogram in Diagnosis and Management.” Colorado Medicine 77(9):320-7, September 1980.
31. Winsor, T. “Influence of Arterial Disease on the Systolic Blood Pressure Gradient of the Extremity.” American Journal of Medical Science 220(2):117-26, 1950.
32. Weir, G.R., Smart, H., vanMarle, H., et al. “Arterial Disease Ulcers, Part 2 Treatment.” Advances in Skin & Wound Care 27(10):462-76, 2014.
33. Sibbald, S.G., Goodman, L., Woo, K.Y., et al. “Special Consideration in Wound Bed Preparation 2011. An Update.” Advances in Skin & Wound Care 24:415-38, 2011.
34. Sibbald, R.G., et al. “Preparing the Wound Bed 2003: Focus on Infection and Inflammation.” Ostomy Wound Management 49(11):24-51, November 2003.
35. Sibbald, R.G., et al. “Increased Bacterial Burden and Infection: The Story of NERDS and STONES.” Advances in Skin & Wound Care 19(8):462-3, October 2006.
36. Lineaweaver, W., et al. “Topical Antimicrobial Toxicity.” Archives of Surgery 120(3):267-70, March 1985.
37. Cooper, M., et al. “The Cytotoxic Effects of Commonly Used Topical Antimicrobial Agents on Human Fibroblasts and Keratinocytes.” Journal of Trauma 31(6):775-84, June 1991.
38. Maklebust, J. “Using Wound Care Products to Promote a Healing Environment.” Critical Care Nursing Clinics of North America 8(2):141-58, June 1996.
39. Maklebust, J., Sieggreen, M. Pressure Ulcers: Guidelines for Prevention and Management, 3rd ed. Springhouse, PA: Springhouse Corp., 2001.
40. Hanz, T., Butler, J.P., Luttrell, T., et al. “The Role of Whirlpool in Wound Care.” Journal of American College of Clinical Wound Specialists 4(1):7-12, 2013.
41. Ramaiah, V., Gammon, R., Kiesz, S., et al. “Midterm Outcomes from the TALON Registry: Treating Peripherals with SilverHawk: Outcomes Collection.” Journal of Endovascular Therapy 13:592-602, 2006.
42. Laird, J.R., Zeller, T., Gray, B.H., et al. “Limb Salvage Following Laser-assisted Angioplasty for Critical Limb Ischemia: Results of the LACI Multicenter Trial.” Journal of Endovascular Therapy 13:1-11, 2006.
43. Bradbury, A., et al. “Final Results of the BASIL Trial (Bypass Verses Angioplasty in Severe Ischaemia of the Leg).” Journal of Vascular Surgery 51(5):1S-76S, May 2010.
44. Dake, M. Zilver PTX Global Data Analysis Highlights DES Benefits in Challenging Patient Populations. Presented at: VIVA, November 6, 2018, Las Vegas, NV.
45. Dake, M.D., Ansel, G.M., Jaff, M.R., et al. Durable Clinical Effectiveness with Paclitaxel-Eluting Stents in the Femoropopliteal Artery: 5-Year Results of the Zilver PTX. Circulation 133(15):1472-83, 2016.

46. Dake, M.D., Ansel, G.M., Jaff, M.R., et al. Sustained Safety and Effectiveness of Paclitaxel-Eluting Stents for Femoropopliteal Lesions: 2-Year Follow-Up from the Zilver PTX Randomized and Single-Arm Clinical Studies. Journal of the American College of Cardiology 61(24):2417-27, 2013.
47. Mousa, A.Y., Beauford, R.B., Flores, L., et al. “Endovascular Treatment of Iliac Occlusive Disease: Review and Update.” Vascular 15(1):5-11, 2007.
48. Schurmann, K., Mahnken, A., Meyer, J., et al. “Long-Term Results 10 Years After Iliac Arterial Stent Placement.” Radiology 224:731-8, 2002.
49. Kedora, J., Hohmann, S., Garrett, W., et al. “Randomized Comparison of Percutaneous Viabahn Stent Grafts vs Prosthetic Femoral-popliteal Bypass in the Treatment of Superficial Femoral Arterial Occlusive Disease.” Journal of Vascular Surgery 45:10-6, 2007.
50. Dalman, R.L. “Long-term Results of Bypass Procedures.” In J.M. Porter, L.M. Taylor, eds. Basic Data Underlying Clinical Decision Making in Vascular Surgery. Annals of Vascular Surgery 141-3, 1995.
51. CAPRIE Steering Committee. “A Randomised, Blinded, Trial of Clopidogrel versus Aspirin in Patients at Risk of Ischaemic Events.” Lancet 348:1329-39, 1996.
52. Hughson, W.G., et al. “Intermittent Claudication: Prevalence and Risk Factors.” British Medical Journal 1(6124):1377-9, May 1978.
53. Clyne, C.A., et al. “Smoking, Ignorance, and Peripheral Vascular Disease.” Archives of Surgery 117(8):1062, August 1982.
54. Margolis, D.J., Kantor, J. “A Multicenter Study of Percentage Change in Venous Leg Ulcer Area as a Prognostic Index of Healing at 24 Weeks.” British Journal of Dermatology 142(5):960-4, May 2000.
55. Coffman, J.D. “Principles of Conservative Treatment of Occlusive Arterial Disease.” in J.A. Spittell, ed. Clinical Vascular Disease. Philadelphia, PA: FA Davis, 1983.
56. The Sage Group. “Diabetic Foot Ulcers, Peripheral Arterial Disease and Critical Limb Ischemia.” Available at: http://thesagegroup.us/pages/reports/dfu-statistics.php
57. Hirsch, A.T., Criqui, M.H., Treat-Jacobson, D., et al. “Peripheral Arterial Disease Detection, Awareness, and Treatment in Primary Care.” JAMA 286:1317-24, 2001.
58. Society for Vascular Nursing. Core Curriculum: Vascular Nursing, 2nd ed. Philadelphia, PA: Wolters Kluwer, 2014.

18

Diabetic Foot Ulcers

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Objectives

After completing this chapter, you'll be able to:

- state the significance of foot ulcers in patients who have diabetes mellitus
- list strategies for preventing foot ulcers in patients with diabetes
- describe wound characteristics and assessment parameters for a patient with diabetes
- list options for reducing pressure for a patient with diabetes who has a foot ulcer
- discuss the therapy options for patients with a diabetic foot ulcer.

We gratefully acknowledge the contributions of Susan van Asten, MD, MSc for her work on previous editions of this chapter.

Diabetes: A Growing Problem

Diabetes mellitus (DM) is a pandemic. The prevalence of diabetes is rapidly increasing globally. In 2015, the world prevalence of diabetes was estimated to be 415 million. By 2040, this is expected to increase to 642 million.[1] In the United States, over the past 30 years, we have continually exceeded the predictions for the prevalence of diabetes. In 1990, the prevalence of diabetes in adults in the United States was 2.5%, affecting 6.2 million people.[2] By 2017, there were 30 million people in the United States with diabetes, afflicting 9.7% of the adult population.[3] The volume of diabetes-related complications is expected to increase as the prevalence of the disease increases.

This chapter on diabetic foot ulcers (DFUs) may be the most important chapter of this text. It represents a disease process that is increasing dramatically. It may change the diagnosis and treatment of ulcers that have been traditionally grouped in different categories like venous stasis ulcers and pressure injuries. The presence of diabetes will fundamentally change the prognosis for these ulcers because the characteristics of the host will be altered.

The diabetic foot syndrome (DFS) includes comorbidities that are caused or exacerbated by diabetes and contribute to the development of foot ulcers, infections, and amputations. The components of the DFS include peripheral sensory neuropathy, peripheral arterial disease (PAD), limited joint mobility, structural foot deformity, abnormal bone metabolism, postural instability, immunopathy, poor nutrition, and Charcot neuroarthropathy.

Diabetic foot complications are one of the most common complications of diabetes that lead to hospitalization.[4,5] Admissions for foot complications account for 20% to 33% of all hospital days for patients with diabetes.[6] The incidence of DFUs is 2% and 7% per year.[6,7] The incidence of ulceration is significantly higher in people with diabetes that have had a previous ulcer or amputation and in patients with dialysis and end-stage renal disease. For instance, the incidence of ulceration increases to 30% to 50% a year in people with an ulcer history.[8,9] Infection is a sentinel event in patients with DFUs

that often leads to hospitalization, amputations, ulcer, and infection recurrence.[10] The incidence of infection in patients with DFUs is 60%.[11,12] Once a patient has been discharged from a hospital for DFU, readmission rate is as high as 50% in the next year.[10]

Box 18-1 ADA Contact Information

The American Diabetes Association (ADA)[2] offers much information for diabetic patients and their families as well as for healthcare professionals. General information about diabetes is available, along with advice on exercise, nutrition, and daily meal planning. Contact: ADA@diabetes.org or 1-800-DIABETES (342-2383).

Diabetes is the most common underlying cause of lower extremity amputation in the United States. Data from the Centers for Disease Control (CDC) report 108,000 diabetes-related amputations or 50 amputations per 10,000 patients with diabetes per year.[13] The risk of lower extremity amputation in people with diabetes is 15 to 46 times higher than in nondiabetic patients.[5] After the initial amputation, the risk of reamputation or amputation of the contralateral extremity is also high: 9% to 17% of patients will experience a second amputation within the same year.[5,14] The incidence of lower extremity amputation is 1.5 times higher in Hispanics and 2.1 times higher in African Americans compared with non-Hispanic Whites.[15] An estimated 2.5 million Hispanic Americans aged 20 years and above have been diagnosed with DM. Between 2004 and 2006, data from different ethnic populations within the United States from ages 20 years and older indicate that 6.6% of non-Hispanic whites, 7.5% of Asian Americans, 10.4% of Hispanics, and 11.8% of non-Hispanic blacks had been diagnosed with DM.[16]

Etiology of and Risk Factors for Foot Ulcers

The risk factors for foot ulceration and amputations are easy to identify from patient history and clinical examination. The highest risk patients to develop an ulcer or amputation are people with a history of previous ulceration or amputation.

The underlying pathology usually is not reversible, and most disease processes affecting the diabetic foot will continue to worsen over time. At its root, DFUs are related to abnormal biomechanics of the foot, limited joint mobility, and sensory neuropathy, so the injury is not perceived by the patient. Three primary pathways or mechanisms of injury have been identified in the development of foot ulcers. These include ulcers that result from ill-fitting shoes (low-pressure injuries that are associated with prolonged or constant pressure), ulcers on weight-bearing areas (repetitive moderate pressure and shear forces on the sole), and penetrating injuries from puncture wounds or other traumatic events (high-pressure injuries with a single exposure of direct pressure).[17] Understanding the etiology is important to plan treatment and to understand prevention strategies to reduce the risk of reulceration.

Peripheral Sensory Neuropathy

Diabetes affects sensory, motor, and autonomic nerve function. In patients with sensory neuropathy, pain, the primary natural warning system that alerts the body to take action, is defective. Sensory neuropathy contributes to an inability to perceive injury to the foot due to what is commonly referred to as loss of protective sensation (LOPS).[18] LOPS represents a level of sensory loss where patients can injure themselves without recognizing the injury. Motor neuropathy contributes to wasting of the intrinsic muscles of the foot; muscle imbalance; and structural foot deformity, such as claw toes and subluxated metatarsophalangeal joints. It is also common for patients with diabetes and neuropathy to have limited range of motion of the ankle and metatarsophalangeal joints. Autonomic neuropathy causes shunting of blood[19] and loss of sweat and oil gland function, which leads to dry, scaly skin that can easily develop cracks and fissures. The combined effect of these neuropathies results in a foot with structural deformity and biomechanical abnormalities, dry skin, and an inability to respond to pain and repetitive injury.

Neuropathy is one of the most common risk factors for lower extremity complications. It is unusual to see a patient with a foot ulcer who does not have sensory neuropathy.[7] Obtaining a complete history is the best way to identify sensory neuropathy.[18,20] Simply asking patients if they have numbness or tingling can identify patients with LOPS.

Early detection of the high-risk foot for patients known to have DM and those not yet diagnosed is essential to reduce complications such as DFUs and amputations. Healthcare professionals need a quick and easy-to-use tool to help screen patients at risk for such complications. Two different screens to assess the feet of patients without diabetes[21] and those known to have diabetes at risk for developing a DFU have been developed and validated by Sibbald and colleagues.[22] (See Table 18-1, Healthy Feet Screen; Table 18-2, 60-Second Screen for the High-Risk Diabetic Foot). Since a person may have diabetes but not have been diagnosed, the Healthy Feet Screen can assist in early detection of skin changes.[21] In one study, when the interprofessional

team implemented the 2012 GENERIC Simplified 60 Second Screen for the High-Risk Diabetic Foot, amputations were reduced by 68%.[22] Ongoing research by Woodbury et al. resulted in adaptations to the original 2012 screening tool in 2015[23] (see Table 18-2).

Table 18-1 Healthy Foot Screen ©2017

Name: _____

DOB (dd/mm/yy): ____/____/____ **Gender: M □ F □**

ID#: _____ **Phone #:** _____

Completed by: _____ **Date of Exam (dd/mm/yy):** ____/____/____

Check both feet and circle responses where applicable

HISTORY	**1. Is the patient known to have diabetes mellitus type 1 or type 2?** *(If yes, perform simplified 60 second screen)*	Yes		No	
	2. Is the patient currently a smoker? *(If yes, counsel on smoking cessation)*	Yes		No	
	3. Does the patient have neuropathy? Ask about burning, stinging, shooting or stabbing in either foot *(Any person with neuropathy-perform simplified 60 second screen)*	**RIGHT** Yes	No	**LEFT** Yes	No
	4. Has the patient had a previous foot ulcer (U) or amputation (A) on either foot? (If yes, *perform simplified 60 second screen)*	Yes U | A	No	Yes U | A	No
INFECTION	**5. Are there signs of toenail fungal infection/inflammatory changes e.g. trauma, psoriasis?** *(If yes, should be treated)*	**RIGHT** Yes	No	**LEFT** Yes	No
	6. Are toe webs abnormal, especially 4/5th toe web? *(If yes, should be treated)*	Yes	No	Yes	No
	7. Is the bottom of the foot dry? (*If yes, should be diagnosed and treated)*	Yes	No	Yes	No
STRUCTURAL CHANGES ***List specific changes in the comments section below***	**8. Is there a significant bony abnormality or associated change?** *(Any significant bony change should trigger a foot specialist referral)*	**RIGHT** Yes	No	**LEFT** Yes	No
CIRCULATION	**9. Can you feel a foot pulse?** (*Absent dorsalis pedis & posterior tibial pulse = vascular laboratory assessment/referral)*	Yes	No	Yes	No
	10. Any pitting edema of the feet/ankle? *If yes, should be diagnosed and treated*	Yes	No	Yes	No

Refer to enabler for more information. Local referral patterns may vary depending on available resources

Other Comments/Abnormalities

_____
_____
_____
_____
_____

Recommendations/Treatment

_____
_____
_____
_____
_____

	ITEMS ON SCREEN	ACTION
1	**DIABETES** **"Yes" if patient is known to have diabetes mellitus type 1 or type 2**	• **Monitor HbA1c** every 3 months • **Perform The simplified 60 second screen**[1] (Accessible at www.WoundPedia.com)
2	**SMOKING** **"Yes" if patient is a current smoker** **Each cigarette:** skin oxygen 30%/1 hr. **Smoking:** host response = delayed healing[2]	☐ **Review smoking cessation options**
3	**NEUROPATHY** **"Yes" if burning, stinging, shooting or stabbing pain on either lower extremity** • **Types of neuropathy:** Sensory, Autonomic, Motor • **Common causes: Diabetes** (neuropathy occurs earlier with poor glycemic control), **B12 deficiency, Trauma, Toxins, Chemotherapy, Cancer, Kidney disease, Idiopathic**	• **Perform The simplified 60 second screen** • **Needs accommodative footwear** • **Optimize HbA1c**
4	**PREVIOUS ULCER OR AMPUTATION** **"Yes" if previous ulcer and/or amputation from history is reported or observed** *Indicates a high-risk foot. Callous = pressure Blister = shear and friction*	• **Perform The simplified 60 second screen** • **Check footwear** • **Refer-Foot specialist** (Chiropodist/Podiatrist)
5	**TOENAIL FUNGAL INFECTION (Onychomycosis)/INFLAMMATORY CHANGES**[3] **"Yes" if nails have distal streaks, white/yellow/brown discolouration, subungal debris** • **Onychomycosis can be suspected if any or all of the above are present** **- 50% of nail disorders are fungus** • **Inflammatory changes e.g. due to trauma, psoriasis, lichen planus**	• **Perform nail clipping** (should include subungal debris) & **confirm diagnosis of onychomycosis with:** - A positive microscopic result for at least one toenail along with clinical presentation AND/OR - A positive culture for fungus • **Treat according to % of nail involvement:** > 60% = Oral Terbinafine 20-60% = Topical Efinaconazole +/ - Oral Terbinafine (> 3 nails) < 20% = Topical Efinaconazole
6	**ABNORMAL TOE WEB** **"Yes" if toe web (particularly 4-5th web space) is dry, scaly (think fungus) OR wet, macerated (think secondary bacterial infection)**	• **Take scraping (dry, scaly) or bacterial swab (wet, macerated)** • **Treat** with antifungals and/or antimicrobials
7	**DRY FOOT** **"Yes" if dryness in a 'Moccasin' distribution (dryness extends to sole, heel & sides of foot)** ☐ **Diagnose cause** (rule out Neuropathy) – Dryness in moccasin distribution– think fungus – Dryness on plantar surface– think neuropathy	• **Take scraping** • **Treat accordingly**
8	**COMMON STRUCTURAL ABNORMALITY** **"Yes" If deformity and/or abnormality in shape or structure of either foot** ☐ **Hammer toe(s)** metatarsal phalangeal joint up, **Claw toe(s)** metatarsal phalangeal joint up + interphalangeal joint down, **Bunion** enlargement of the base of large toe (Hallux Valgus)	• **Check footwear** • **Refer to foot specialist** (chiropodist/podiatrist)
9	**ARTERIAL DISEASE** **"Yes" if any of the pedal pulses are absent** • Palpate Dorsalis Pedis, if absent check for Posterior Tibialis • **Clinical signs:** cold feet, decreased capillary refill, dependent rubour, intermittent claudication	☐ **If both dorsalis pedis and posterior tibialis pulses are absent = vascular laboratory assessment and/or referral**
10	**VENOUS DISEASE** **"Yes" if pitting edema of lower legs, feet/ankle is present** • Associated with varicosities, hyperpigmentation, lipodermatosclerosis (hard woody change) • If the hard skin extends to the toes and the skin over the second toe cannot be pinched = + ve Stemmer's sign = Lymphedema	• **Diagnose cause and prescribe compression therapy** if no arterial compromise • **Consider Pentoxifylline (Trental)** • **Avoid long periods of standing or sitting** • **Elevate legs and calf-muscle pump exercises**

[1] *Woodbury MG et al. Tool for Rapid & Easy Identification of High Risk Diabetic Foot: Validation & Clinical Pilot of the Simplified 60 Second Diabetic Foot Screening Tool. PLoS ONE. 2015;10(6):e0125578. doi:10.1371/journal.pone.0125578,* [2] *Jensen JA. Cigarette smoking decreases tissue oxygen. Arch Surg. 1991;126(9):1131,* [3] *Gupta AK et al. Canadian Approach with a New Transungual Treatment: Development of a Clinical Pathway. J Cutan Med Surg. 2015 Sep-Oct;19(5):440-9*

OTHER ABNORMALITIES/LESIONS:

- **Psoriasis:** Discrete red plaques + silvery scale = look elsewhere, Sterile yellow (new) to brown pustules especially in females that smoke
- **Dermatitis- Nummular**: Annular lesions/ dorsum; **Contact**: Allergic – dorsum, irritant –plantar
- **Cellulitis:** Bacterial infection – painful red warm dermal swelling ☐ **Callus:** Hyperkeratosis over pressure points
- **Corn or clavus**: Often small; **Hard corn**: small hyperkeratotic spot – on plantar skin; **Soft Corn**: often in the 4th & 5th web space
- **Gouty Tophus:** Painful lesion often on the large toe associated with a white chalk like discharge
- **Morton's Neuroma**: Inflamed plantar nerve, tender nodule at the base of 3-4th toes
- **Plantar Wart:** Has keratotic surface with punctate bleeding points that pushes away skin surface markings
- **BENIGN: Stucco keratosis (SK)**: aging spot (small seborrheic keratosis = SK); **Melanocytic nevus**: pigmented lesion with normal nevus cells; **Dysplastic nevus:** larger size and more variable colour
- **PRECANCEROUS: Actinic keratosis (AK):** red, rough, scaly spot, common in areas of sun exposure; **Cutaneous horn**: thin hyperkeratotic column with variable base e.g. AK + SK, or skin cancer (SCC, Other)
- **SKIN CANCER: Basal Cell Carcinoma**: translucent papules, ulcers with telangiectasia associated with the base; **Squamous Cell Carcinoma**: irregular keratotic papule that may be painful; **Malignant Melanoma**: asymmetry, irregular border, colour variability- black, blue, red, white, > 6mm and evolving

Table 18-2 60-Second Foot Screening Tool©

Table 2	GENERIC- Simplified 60 Second Screen for the HIGH RISK DIABETIC FOOT 2016				©Sibbald (2012)
Name: _____ **ID#:** _____ **Phone #:**_____ **Facility:**_____ **DOB (dd/mm/yy):**_____/_____/_____ **Gender: M ☐ F ☐ Years with diabetes:**_____ **Ethnicity: Black ☐ Asian ☐ Caucasian ☐ Aboriginal ☐ Other ☐** **Date of Exam (dd/mm/yy):**_____/_____/_____		**CHECK BOTH FEET** (Circle correct response) **YES on either foot is = HIGH RISK**			
		LEFT		**RIGHT**	
History	**1.** Previous Ulcer	NO	YES	NO	YES
	2. Previous amputation	NO	YES	NO	YES
Physical exam	**3. Deformity**	NO	YES	NO	YES
	4. Ingrown toenail (thickened nail fold)	NO	YES	NO	YES
Foot lesions *Remember to check 4th and 5th web spaces and nails for fungal infection Remember to check for inappropriate footwear*	**5.** Absent pedal pulses (Dorsalis Pedis and/or Posterior Tibial)	NO	YES	NO	YES
	6. Active ulcer	NO	YES	NO	YES
	7. Blisters	NO	YES	NO	YES
	8. Calluses (thick plantar skin)	NO	YES	NO	YES
Neuropathy *MORE THAN 4/10 SITES LACKING FEELING =* ***"YES"***	**9.** Monofilament exam **(record negative reaction):** **a)** Right_____/10 negatives (≥4 negatives = Yes)	NO	YES	NO	YES
	b) Left_____/10 negatives (≥4 negatives = Yes)	NO	YES	NO	YES
		Total # of YES:____		**Total # of YES:**____	

PLAN

a) **POSITIVE SCREEN-** Results when there are one or more "Yes" responses. **Refer to a foot specialist or team for prevention, treatment and follow up**. (Bony deformity, current ulcer, absent pulse are most urgent). These individuals are at increased risk of a foot ulcer and/or infection. Patients should be educated on what changes to observe and report, while waiting for the specialist appointment.

Referral to: _____ **Appointment time:**_____

b) **NEGATIVE SCREEN-** Results when there are all "No" responses. **No referral required.**
Educate patient to report any new changes to their healthcare provider and re-examine in 1 year.

One Year Date for Re-Examination (dd/mm/yy):_____/_____/_____

Completed By: _____ **Date:** _____

Additional Note:

For **POSITIVE SCREENING**, in addition to referral plan above, **positive risk factors** can be linked to the care recommendations in "Root Risk Classification and Follow-Up Guide" table on the bottom of reverse side. Local referral patterns may vary depending on expertise and available resources.

GUIDELINES FOR USE: © Sibbald (2012)

GENERIC-Simplified 60-Second Screen for the HIGH-RISK DIABETIC FOOT 2016

General Instructions:

This diabetic foot screening tool is designed to identify individuals with high-risk diabetic feet. The screening tool is a simplified 60-second assessment for each foot, to be implemented by any healthcare provider. Preparation involves having a 5.07g monofilament available and asking patient to remove their shoes and socks.

Normal screening findings are indicated as "No" (not requiring referral) and abnormal screening findings are indicated as "Yes" (requiring referral). Generation of a list of local reputable foot specialists and/or teams for referring is recommended.

Screening involves:

- Inform patient about the simplified 60-second screening and explain the reason for the examination.
- Fill in patient's demographic data in top left section of screening tool.
- Assess both feet. Circle either a "Yes" or "No" response to questions 1-9.
- **Any "Yes" response requires follow up or a referral to a foot specialist and/or team.**

Question	"Yes" Response
1	**"Yes", if previous ulcer from history is observed:** Ask the patient and assess both lower legs and feet for the presence of a healed ulcer as evidenced by scar tissue.
2	**"Yes", if previous amputation:** of digit(s), foot or limb is observed.
3	**"Yes", if deformity:** and/or abnormalities in shape or structure of either feet is observed (bony prominences/hammer toes).
4	**"Yes", if ingrown toenail present.** Inspect distal corners for embedded nail and/or thickened nail fold skin.
5	**"Yes" If absent pedal pulses** (palpate Dorsalis Pedis and if absent check Posterior Tibial). A yes answer requires absence of both pulses.
6	"**Yes", if active ulcer(s) present:**Openings in the skin with a dermal or deeper base.
7	**"Yes", if blisters present:** Observe for fluid (serum, blood or pus) under intact skin surface.
8	**"Yes", if callus present (thick scale on plantar skin):** Assess and inspect for presence of thick areas of keratin on the bottom or sides of feet and toes.
9	**"Yes" of monofilament Exam identified 4 or more negative reactions (lack of feeling)**: Follow the monofilament exam instructions below. Each foot is examined separately.

Steps for Monofilament Test for Neuropathy:

- Show and touch monofilament to patient's arm or upper leg.
- Ask the patient to close their eyes and say yes when they feel the monofilament.
- Touch monofilament until filament bends in a letter "c" shape, assessing all 10 areas on diagram (Do not test over calluses, scars or ulcers)
- **Lack of feeling (4 or more out of 10) - indicates a negative reaction = Neuropathy = "YES" on screening tool**

Foot Risk Classification and Follow-up Guide

Assessment Findings ↓	Risk	Follow Up (mths)	Prof. Nail Care	Shoes	Orthotics + Diabetic Socks	Activity
No Neuropathy	0	12	-	Well fitting	Well fitting shoes	As able
Neuropathy	1	6	+/-	Professional fit	Custom full contact	As able, monitor, guided by foot exam
Deformity	2a	3-4	+/-	+/- custom fit	Custom full contact	Avoid excessive walking, non-impact exercis e
Peripheral Vasuclar Disease	2b	3-4	+	Professional fit	Softfull contact	Dependent on ischemic pain, non-impact exercis e
Ulcer Hx or Active ulcer	3a	1-2	+/-	Professional fit	Custom fitted	Activity dependant on exam, non-impact exercises
Hx Amputation	3b	1-2	+/-	Special clinic (assessment) Modified footwear	Specialized clinic: amputation/prosthess, +/- walking aid	Based on tissue tolerance, non-impact exercises

Modified from International Diabetes Federation, International Working Group on the Diabetic Foot 2007

Several screening methods can be used to identify sensory neuropathy, including a systematic clinical examination, vibration perception threshold (VPT) testing with a VPT Meter, and pressure assessment with Semmes-Weinstein monofilaments[24] (Fig. **18-1** A and B). These methods are inexpensive, quick to do, and noninvasive; can be performed by almost anyone; and have good sensitivity and specificity to identify patients with LOPS.[20] All of the testing procedures should be demonstrated on the hand or arm of the patient, so they have a better idea of what the stimulation should feel like at a site that is more likely to have normal sensation.[25,26]

VIBRATION PERCEPTION THRESHOLD METER
On-Off
Voltage
Min.
Max.
XILAS MEDICAL, INC.
A

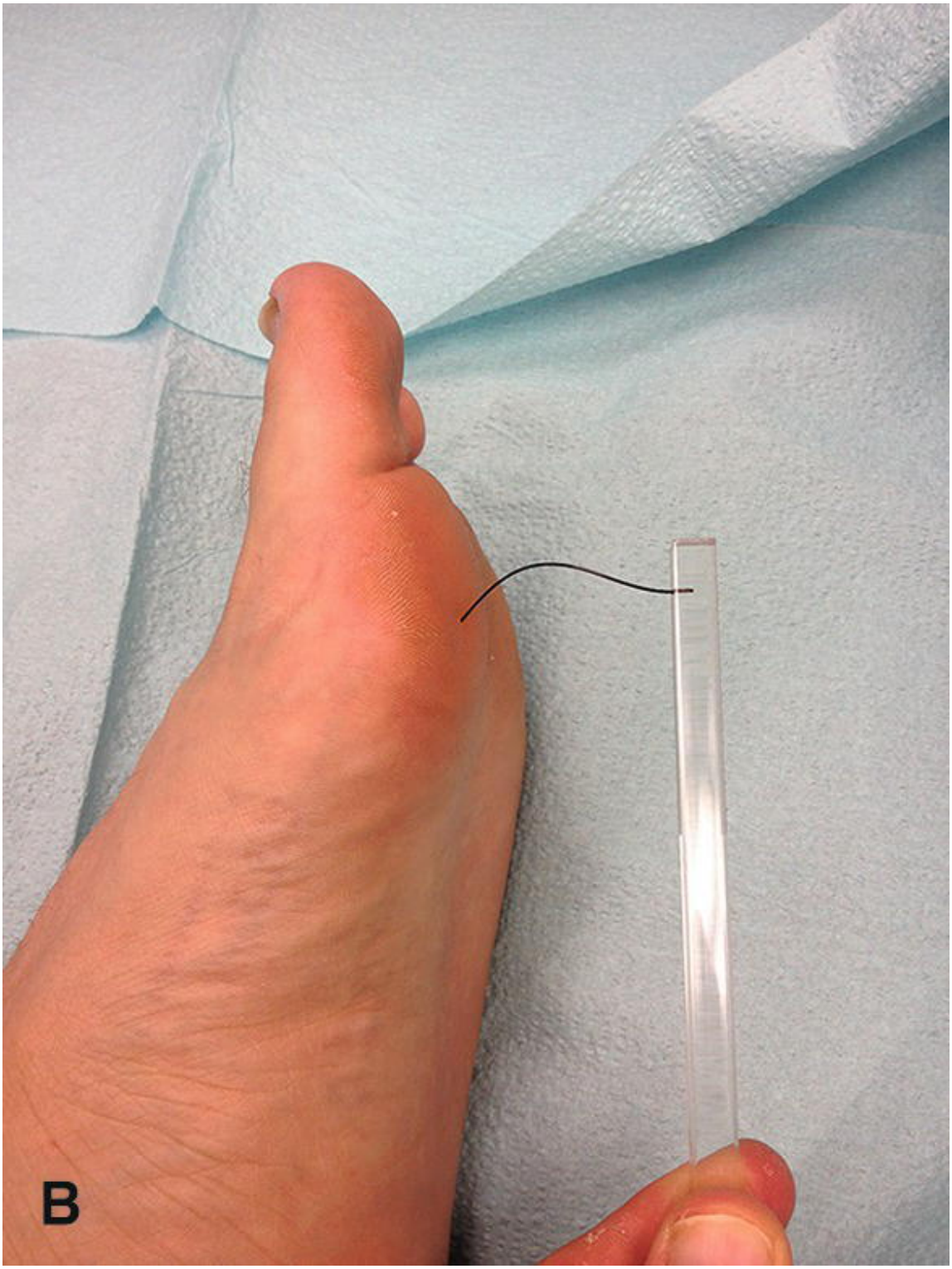


Figure 18-1. (A) VPT Meter. **(B)** Semmes-Weinstein monofilament. (Photo courtesy of S. van Asten and J. Lafontaine, MD; used with permission.)

Vibration perception is one of the easiest tests to evaluate large nerve fiber loss. Using a 128-Hz tuning fork, strike the tuning fork on the heel of your hand, so you hear an audible clang. Then apply the end of the tuning fork to a bony prominence like the head of the first metatarsal. Ask the patient to tell you if he or she feels the vibration and then when the vibration stops. Patients without sensory neuropathy should feel vibration for more than 15 seconds[26] (Fig. 18-2).

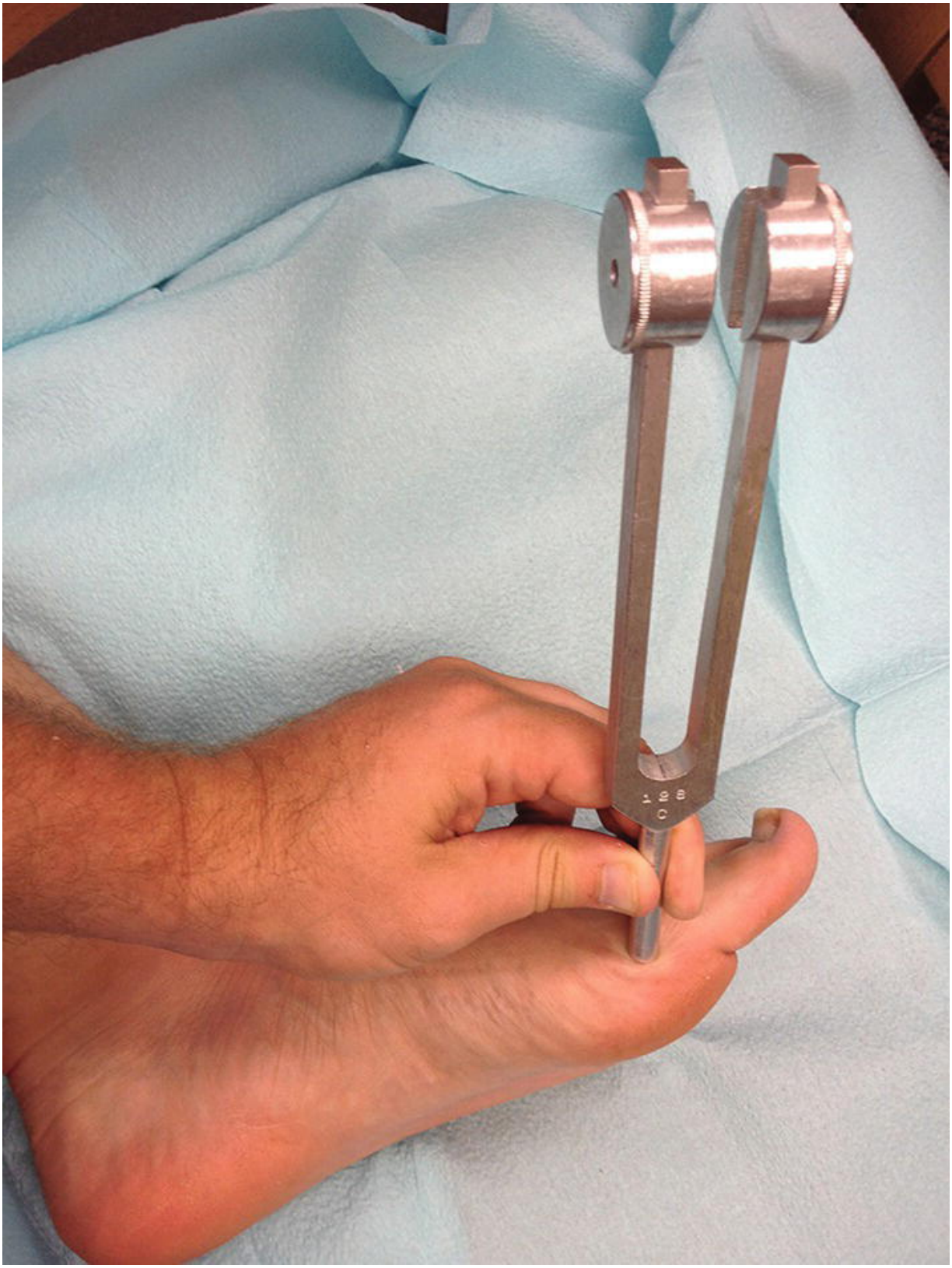


Figure 18-2. Tuning fork exam over bony prominence. (Photo courtesy of S. van Asten and J. Lafontaine, MD; used with permission.)

VPT testing is a quantitative evaluation that measures large myelinated nerve function. The VPT device (Fig. 18-1A) is expensive, and it is not especially portable, so it is best suited for neuropathy evaluation in a clinic setting. It is very easy to use, and it provides a quantitate measure that makes interpretation easier. It is less prone to interoperator variation than monofilaments, and it does not need to be replaced to continue providing accurate results. The VPT Meter is a handheld device with a rubber head that is applied to a bony prominence, such as the medial aspect of the first metatarsal head or the tip of the great toe. The unit contains a linear scale that displays the applied voltage, ranging from 0 to 100 V. The amplitude is then slowly increased until the patient can feel the vibration. The inability to feel greater than 25 V is indicative of LOPS and puts the patient at risk for ulceration and amputation.

The Semmes-Weinstein monofilament (Fig. 18-1B) is a sensory assessment tool. This assessment tool consists of a set of monofilaments that vary in thickness and diameter; the gradient forces of these monofilaments range from 0.086 to 448 g. These monofilaments are used to map out sensory loss. The results are useful in comparing the sensory loss with the return of sensations on various regions of the body. Semmes-Weinstein monofilaments should be purchased from a vendor that sells *calibrated* instruments because considerable variability exists among different brands of monofilament.[17] Lavery and Booth found that some brands of monofilaments buckled at 8 g of force rather than at the 10 g for which they were designated.[27,28] In addition, the material properties of the monofilament wear out after repetitive testing. Yong et al.[29] found that after 500 cycles of testing (or the equivalent of testing 10 sites on each foot for 25 patients), there was an average reduction of 1.2 g of testing force. A worn-out monofilament may result in patients being diagnosed as having sensory neuropathy with LOPS when they are not at risk.

Practice Point

A Semmes-Weinstein 10-g (5.07 log) monofilament is commonly used to assess protective sensation in the feet of patients with diabetes.[27–29]

Use the 10-g (5.07 log) monofilament wire on each foot at the following 10 sites:

- plantar aspect of the first, third, and fifth digits
- plantar aspect of the first, third, and fifth metatarsal heads
- plantar midfoot medially and laterally
- plantar heel
- dorsal aspect of the midfoot.

Performing the Test

Place the patient in a supine or sitting position with bare feet, toes pointing straight up. Touch the monofilament to the patient's arm or hand to demonstrate what it feels like. Then ask the patient to respond "yes" each time he or she feels the monofilament on the foot (Fig. 18-3).

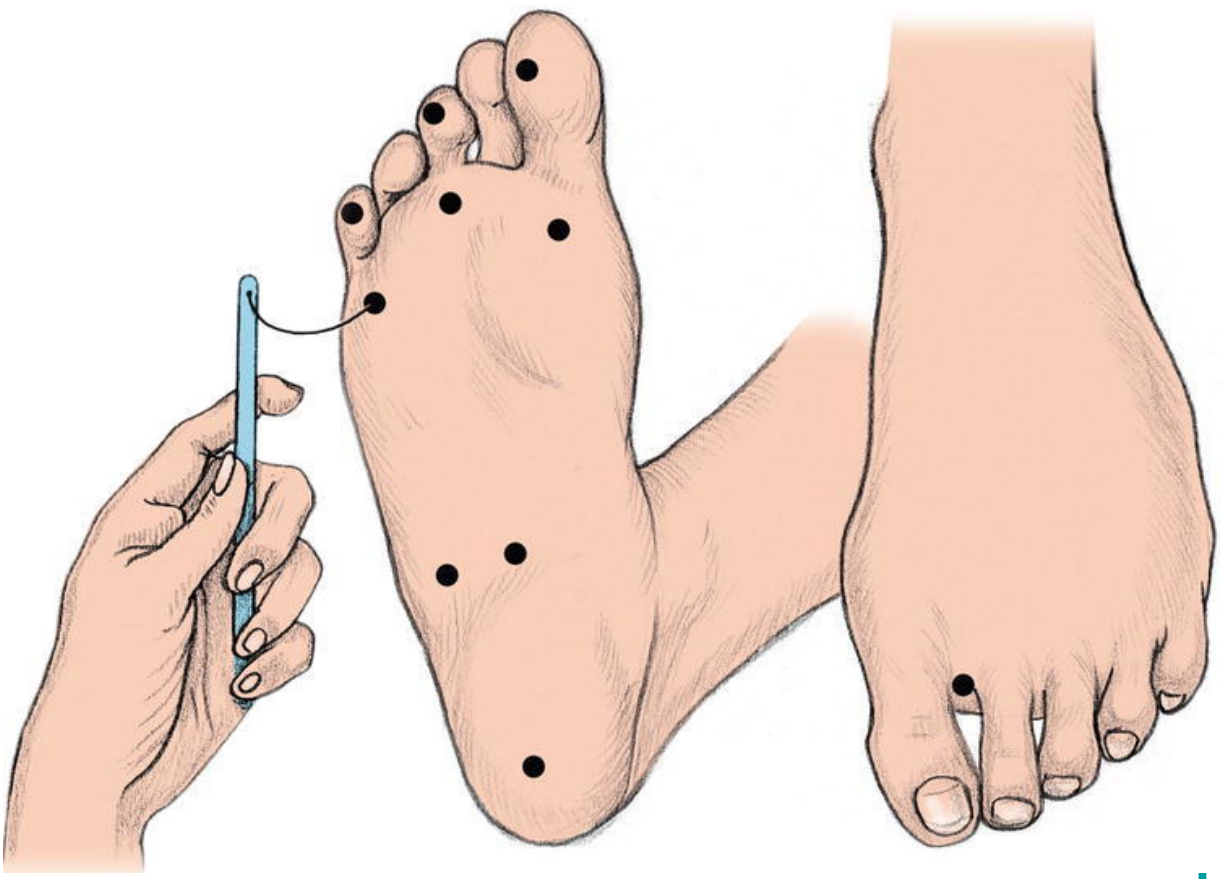


Figure 18-3. Assessing protective sensation with a monofilament.

Ask the patient to close the eyes. Remind the patient to say “yes” when he or she feels the monofilament on the foot; *do not ask the patient*, “Do you feel this?” as you place the monofilament on each of the 10 sites on each foot. (*Even if the patients do not feel the monofilament, they could answer “yes” to your questions*.) Hold the monofilament perpendicular to the patient’s foot, and press it against the first site, increasing the pressure until the monofilament wire bends into a C shape. Make sure it doesn’t slide over the skin. Hold the monofilament in place for about 1 second. Record the patient’s response on a foot screening form. Then move to the next site.

Test all 10 sites at random, and vary the time between applications so that the patient won’t be able to guess the correct response. If the patient has a scar, callus, or necrotic tissue at a test

site, apply the monofilament along the perimeter of the abnormality, not directly on it.

LOPS is indicated if the patient can't feel the monofilament at any site on the foot. It is essential to teach a patient who has lost protective sensation to inspect and protect his feet.

Practice Point

Not all monofilaments are of the same quality or last forever; be sure to use a calibrated instrument in your patient assessments.

Peripheral Arterial Disease

Tissue perfusion is arguably the most important aspect of diabetic limb salvage. PAD in patients with diabetes is characterized by multiple occlusive plaques of small- and medium-sized arteries of the infrapopliteal vessels.[30,31] Hence, diabetes-induced PAD affects both small and large vessels. This becomes an important consideration for interpreting the results of arterial diagnostic tests as well as developing a treatment plan. PAD puts the patient with diabetes at a significantly greater risk for foot ulcers, infections, and amputations.[17] Several theories attempt to explain the microvascular changes that occur in diabetes. One theory proposes that increased microvascular pressure and flow results in direct injury to the vascular endothelium, which in turn causes the release of extravascular matrix proteins. This leads to microvascular sclerosis and thickening of the capillary basement membrane. Capillary fragility also leads to microhemorrhage, which could be the reason that infection spreads through the tissue planes in patients with diabetes.[31] In addition to the direct effect on the vessels, an additional indirect effect on the microvasculature is mediated by the autonomic nervous system. LoGerfo and Misare[32] believe that there is no microcirculatory occlusive process; rather, they suggest that some other indirect physiologic abnormality occurs. Altered microvascular blood flow is a complication of diabetic autonomic neuropathy that causes a shunting of blood away from the skin, making it prone to ulceration and impairing the healing process[31] (Fig. 18-4).

Figure 18-4. Focal necrosis of the third toe. (Photo courtesy of S. van Asten and J. Lafontaine, MD; used with permission.)

Evaluating vascular status should include a thorough history of symptoms of intermittent claudication, ischemic rest pain, and peripheral vascular surgery; clinical signs of ischemia, such as skin temperature, dependent rubor, pallor, hair loss, and shiny skin; and a clinical assessment of lower extremity pulses.[33] According to the American College of Cardiology (ACC)/American Hospital Association (AHA) guidelines[34] for the management of patients with PAD, physicians should screen their patients for a diagnosis of PAD by determining bilateral resting ankle–brachial indices (ABIs) (Fig. 18-5). In addition, a toe–brachial index should be used when the ABI is not reliable because of excessively high systolic pressures in the ankle.

When the ABI is greater than 1.3, the arteries are considered to be noncompressible due to calcifications within the endothelial walls. Segmental pressure measurements are useful to localize the site of lower extremity PAD when planning a vascular intervention. However, in the advanced disease state of diabetes and particularly end-stage renal disease, ABIs may have limited utility due to the lack of compressibility and may require vascular surgery consultation.

Bidop

Figure 18-5. Handheld Doppler arterial exam of posterior tibial artery. (Photo courtesy of S. van Asten and J. Lafontaine, MD; used with permission.)

Skin and Nail Examination

Evaluation of the skin and nails is critical to identify the subtle signs of impending injury, including high-pressure areas, cracks, maceration, blistering, or fissures in the skin. Patient education is an important aspect of care. All patients should be instructed on how to perform skin self-examinations as a preventive measure. Discoloration of a callus or bleeding under a callus is a sign of a preulcerative lesion. Likewise, deformed and thickened nails are commonly the source of abnormal pressure on the nail bed that can cause subungual ulcerations. Common nail disorders seen in patients with DM include onychomycosis (tinea unguium) and onychocryptosis (Fig. 18-6). While these are usually minor problems in persons without diabetes, they can result in cellulitis, osteomyelitis, neuropathy, and vascular impairment in patients with diabetes.

Figure 18-6. Ingrown toenail. (Photo courtesy K. Zinszer, DPM; used with permission.)

Musculoskeletal Examination

In patients with neuropathy, ulcerations typically develop as a result of repetitive pressure and shear on the sole of the foot or from shoe pressure on the top or sides of the foot; however, no specific level of pressure has been determined to be abnormal or pathologic.[35] Diabetes alters biomechanics in patients with preexisting structural and functional foot deformities (Fig. 18-7). Motor neuropathy is thought to contribute to atrophy and weakness of the intrinsic muscles of the foot. This leads to what has been called the “intrinsic minus foot,” which describes wasting of the small (intrinsic) muscles that originate in the foot (flexor digitorum brevis, flexor hallucis brevis, extensor

digitorum brevis, extensor hallucis brevis, lumbricales, interossei, and abductor hallucis).

Figure 18-7. Ulcer under two to three metatarsophalangeal joints after great toe amputation. The two to three metatarsophalangeal joints are dislocated, causing increased pressure on the sole of the foot. (Photo courtesy of S. van Asten and J. Lafontaine, MD; used with permission.)

Metatarsal ulcers can develop when digital deformities are irritated by the toe box of a shoe or because of loss of fat pad and increased pressure under the metatarsal head (Figs. 18-8 and 18-9). The lesser digits contract and sublux dorsally, resulting in a claw toe deformity and a strong plantar flexor force at the metatarsophalangeal joints.[36] As the toes deform and the metatarsophalangeal joints dislocate, the metatarsal heads are literally driven through the bottom of the foot. The tips and dorsal aspects of the toes and the area beneath the metatarsophalangeal heads are subjected to increased pressure and friction, which, in the presence of LOPS, can lead to ulceration.[37] Limited mobility of the ankle and metatarsophalangeal joints has been associated with soft tissue glycosylation involving the gastroc–soleus–Achilles complex and periarticular tissues.[38] Limited motion of the ankle, subtalar, and metatarsophalangeal joints has been associated with high pressures in the forefoot. Often patients with an intrinsic minus foot will appear to have a high arch; however, this is not a congenital deformity but rather is due to atrophy of the abductor hallucis muscle belly on the medial side of the foot. A profound example of musculoskeletal abnormality of the diabetic foot is represented by Charcot neuroarthropathy (Fig. 18-10) which is characterized by fracture, subluxation, and/or dislocation of joints in the foot or ankle. These structural deformities typically cause plantar bony deformities resulting in areas of high pressure leading to ulceration.

Figure 18-8. Metatarsal ulcer. (Photo courtesy of S. van Asten and J. Lafontaine, MD; used with permission.)

Figure 18-9. Ulcer on digital deformity. (Photo courtesy of S. van Asten and J. Lafontaine, MD; used with permission.)

Figure 18-10. Charcot deformity. (Photo courtesy of S. van Asten and J. Lafontaine, MD; used with permission.)

Infection

Soft tissue and bone infections are very common in patients with diabetic foot ulcerations. The majority of patients with DFUs (56%) will be treated for soft tissue infection during the course of their ulceration. Approximately 20% of these patients will develop infection of the underlying bone.[39] Classically defined signs and symptoms and infection classification are outlined in the Infectious Disease Society of America (IDSA) guidelines.[40]

Identification of foot infections in patients with diabetes requires vigilance because the normal signs of infection may be blunted or absent. Hyperglycemia impairs the humoral innate immune response by increasing the proinflammatory cytokine levels; dysregulating vasoactive cytokines, such as bradykinin and nitric oxide (NO); and decreasing complement activation. This, in turn, can lead to increased insulin resistance through several pathways, causing more hyperglycemia.[41,42] The polymorphonuclear cells (PMNs) and monocytes of the cellular innate immune system show impaired chemotaxis, adherence, phagocytosis, and intracellular killing in patients with diabetes.[43,44] The influence of hyperglycemia on the adaptive immune system (i.e., T cells and immunoglobulins) is less well understood.

The lower extremity wounds are often colonized with microorganisms regardless of the presence of a “true infection.” Routine cultures of the wounds and superficial swabs should not be done when the wound is not clinically infected.[45] When cultures are obtained, deep tissue should be obtained rather than superficial swabs. In cases of mild or moderate infection, curettages from the ulcer base after debridement should be obtained prior to the initiation of antibiotics.[46] In severe infections, empiric antibiotic therapy should be started as soon as possible. Bone biopsy, preferably under radiographic guidance, is the most reliable method to obtain bone cultures in patients suspected of having osteomyelitis.[47] (See Chapter 7, Wound Bioburden and Infection.) Wound depth is the strongest predictor of both soft tissue and bone infections. Compared with superficial wounds, the risk of infection for wounds that extend to the

bone is 23 times higher for soft tissue and 6.7 times higher for bone (see Table 18-3).

Table 18-3 Risk Factors for Soft Tissue and Bone Infections

	Relative Risk	95% Confidence Intervals	*P* Value
Risk Factors for Soft Tissue Infection			
Wound penetrates to bone	23.08	8.47–62.92	0.0001
Previous history of ulceration prior to enrollment	2.15	1.07–4.32	0.03
Recurrent/multiple wounds during study period	1.92	1.20–3.06	0.007
Risk Factors for Bone Infection			
Ulcer depth to bone	6.71	2.27–19.85	0.001
Ulcer duration >30 d	4.66	1.62–13.37	0.004
Recurrent foot ulceration	2.41	1.28–4.53	0.006
Traumatic etiology to ulcer	2.36	1.12–4.98	0.02
Peripheral vascular disease	1.93	1.04–3.56	0.04

Adapted from Lavery, L.A., Armstrong, D.G., Wunderlich, R.P., et al. Risk Factors for Foot Infections in Individuals with Diabetes. *Diabetes Care* 29(6):1288, June 2006; Lavery, L.A., et al. Risk Factors for Developing Osteomyelitis in Patients with Diabetic Foot Wounds. *Diabetes Research and Clinical Practice* 83(3):347-52, March 2009.

Biofilm has been associated with the failure of wounds to heal; however, there is very little literature to support a high prevalence of biofilm in chronic wounds or that biofilm has a strong association with nonhealing DFUs. Biofilm is a significant growing concern that is less understood, its impact underappreciated, and not effectively treated. Biofilm is a colony of bacteria, fungus, or yeast that can populate a wound within 10 to 24 hours.[48,49] Once established, a complex and biodiverse community evolves protected by a glycocalyx shell.[50,51] The prevalence of biofilm in small studies (n = 15 to 100) in chronic wounds is 23% to 60%,[52,53] so biofilm is not present in all chronic wounds and often not in the majority of chronic wounds. There are no data about the prevalence of biofilm in diabetic foot infections. There is no work that shows that the prevalence of biofilm is different in DFUs that healed and DFUs that do not heal. There is no published work

that shows a change in biofilm when “antibiofilm treatments” are provided or that the change is related to healing or the prevention of infection. There is in fact very little evidence in diabetic wound healing in humans concerning biofilm.

Diagnostic Imaging

Whenever there is an open wound on the foot, it is always wise to order an x-ray initially to check for the presence of osteomyelitis. If the wound has been open for several weeks or if bone can be palpated with a sterile probe, further investigation is warranted even if standard x-rays are negative. The gold standard to diagnose osteomyelitis is bone biopsy with bone culture and histology of the bone. Bone biopsies are easy to perform and no more invasive than a puncture with a 16-gauge needle. In addition, bone cultures provide a definitive diagnosis, and culture sensitivity results can be used to direct antibiotic therapy.

Multidisciplinary Framework for Prevention

Evaluation of risk factors and risk stratification is important to prioritize the patient's treatment according to their individual needs.[54] Many healthcare providers either never evaluate the feet or generally consider everyone with diabetes to be "at risk" for foot problems. This usually leads to no preventive care, but it can also contribute to unnecessary services for low-risk patients. To help evaluate individual risk factors, a risk classification system endorsed by the International Working Group on the Diabetic Foot[7] provides a validated scheme to stratify subjects based on their risk of ulceration and amputation. Key elements of the lower extremity examination should help to risk-stratify subjects in order to identify the frequency and level of preventive care required (Table 18-4).

Table 18-4 Lavery-Peters Diabetic Foot Risk Classification

Category	Risk Factors	Ulcer Incidence	Amputation Incidence	Prevention and Treatment
1	No neuropathy No peripheral arterial disease (PAD) No history of ulcer	2%	0.04%	• Reevaluation once a year
2	Neuropathy ± deformity No PAD No history of ulcer or amputation	3.4%	0.05%	• Podiatry every 6 mo • Over-the-counter shoes and insoles; evaluate appropriate fit
3	PAD No history of ulcer or amputation	13.8%	3.7%	• Podiatry every 2–3 mo • Professionally fit therapeutic shoes and insoles • Patient education
4	Previous ulcer or amputation	31.5%	8.1%	• Podiatry every 1–2 mo • Professionally fit therapeutic shoes and insoles • Patient education

Adapted from Lavery, L.A., Peters, E.J., Williams, J.R., et al. Reevaluating the Way We Classify the Diabetic Foot: Restructuring the Diabetic Foot Risk Classification

System of the International Working Group on the Diabetic Foot. *Diabetes Care* 31(1):154-6, January 2008.

Many strategies can be used to help prevent foot complications in diabetic patients. Several clinical studies have reported a 48% to 78%[55] reduction in amputations and a 47% to 49% reduction in lower extremity–related hospitalizations[56] when high-risk diabetic patients are treated in specialty clinics. These clinics often include multiple specialties that focus on both prevention and care of acute complications in patients with diabetes. Documents for prevention measures related to the "diabetic foot" have been developed by the American Orthopaedic Foot and Ankle Society[57] and the American College of Foot and Ankle Surgery.[58]

Multidisciplinary teams are an important principle in the comprehensive care of high-risk patients with diabetes. A "spoke and hub" model for diabetic limb salvage utilizes community-based wound care centers to screen, assess, diagnose, and treat the majority of wounds while referring the most difficult cases to tertiary referral centers within a regional area where a more resource intensive approach can be utilized. Thus, a distinction between a "wound care center" (community based) and a "limb salvage center" (tertiary referral center) should be used.[59] This approach allows a dedicated diabetic limb salvage team to treat a high volume of the highest risk patients.

Another evolution of wound care thought may include a period of intensive inpatient care. Currently, weekly or biweekly clinic-based treatment is the norm that may result in little demonstrable progress. An inpatient course that includes tight glucose control/Hemoglobin A1C, nutrition optimization, perfusion maximization, intravenous antibiotic therapy, and serial operating room wound debridement with a plan for definitive wound closure or coverage may be of benefit.

Further complicating the treatment of chronic wounds is the diversity of wound care providers. Wound care is a discipline that spans multiple disciplines. Thus, wound treatment is driven by the scope of training, licensing, and differing philosophical approaches. This is another reason for collocated wound care where the strengths

of each discipline can be combined to a single, comprehensive treatment approach.

Foot Care

Regular foot evaluation is essential to identify new risk factors and prevent impending complications. Podiatric physicians debride callus and nails as well as regular evaluation of the patient's shoes and insoles. These routine encounters offer an additional opportunity to reinforce key educational elements, such as the need to avoid going barefoot, hydrate the skin, and inspect the feet daily. Protective footwear and insoles can be prescribed for the patient and then evaluated and monitored for their effectiveness.

The Diabetic Shoes and Insoles

Patients with diabetes who are "at risk" for foot disease and who have Medicare Part B are eligible for Medicare's Therapeutic Shoe Bill.[60] In order to qualify, a patient must have diabetes and one or more of the following: previous amputation of part or all of either foot; a history of previous foot ulceration or preulcerative calluses; peripheral neuropathy with evidence of callus formation; foot deformity; or poor circulation.

The Medicare Part B (medical insurance)[60] bill covers the furnishing and fitting of either of these each calendar year, if you have diabetes and severe diabetic foot disease:

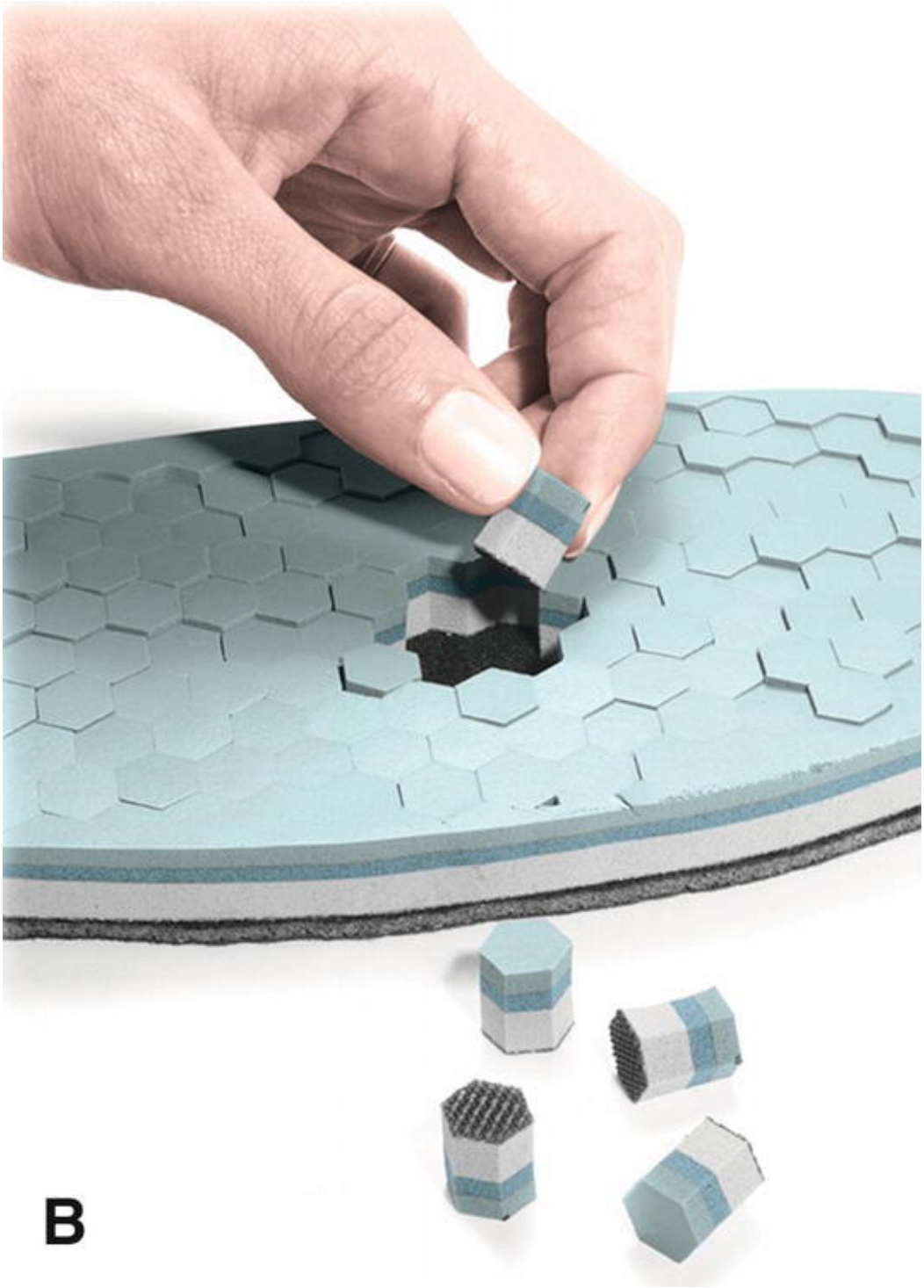
- One pair of custom-molded shoes and inserts
- One pair of extra-depth shoes

Medicare also covers:

- Two additional pairs of inserts each calendar year for custom-molded shoes
- Three pairs of inserts each calendar year for extra-depth shoes

Medicare will cover shoe modifications instead of inserts[60] (Fig. 18-11A–C).

A



B

Figure 18-11. (A) Wide-toe shoe with insert. **(B)** Shoes with a deep toe box and that are “extra depth” throughout the shoe are the mainstay of diabetic wound preventive care. **(C)** The shoes typically come with laces, as shown here, or with Velcro closures. The combination of a correctly sized shoe and an accommodative insole can reduce pressure on the sole, top, and sides of the foot. The insert is customized to relieve pressure, according to the patient’s needs, and then placed inside the shoe. (Photographs courtesy of Royce Medical Co.)

Protective Footwear and Pressure Redistribution

The primary role of therapeutic footwear and insoles is to protect the foot from repetitive injuries and eliminate the shoe as a source of pathology. Extra-depth shoes have a high toe box with enough depth throughout the shoe to accommodate a total contact molded insole or orthotic. These are often recommended for patients with structural foot deformities, such as claw toes or dislocated metatarsophalangeal joints. These types of shoes usually allow for up to a ⅜″-thick accommodative insole to fit without irritating the top or sides of the foot (Fig. 18-12).

Figure 18-12. Hemisection of diabetic extra-depth shoe with insole.

The combination of a correctly sized shoe and a protective insole can reduce pressure on the sole, top, and sides of the foot by as much as 20%.[61,62]

There are a variety of insole materials and material combinations as well as different accommodations that can be built into the insole. Likewise, the types of shoe and outer sole accommodations are numerous. Most of the decisions for protective shoes and insoles are left to technicians that have little working knowledge of the medical literature. When patients reulcerate, they do not return to the pedorthist or shoemaker for care, so these providers have no follow-up to determine if their approach is effective.[62,63] Custom-molded shoes (Figs. 18-11 to 18-13) are individually made from a mold of the patient's foot. Custom-molded shoes can be expensive and require several weeks or months to make. However, they are only necessary in high-risk patients with severe foot deformities that cannot be accommodated by off-the-shelf shoes. For most patients with less severe deformities, there are a number of more affordable athletic, comfort, and therapeutic shoes with multiple sizes and extra depth to accommodate a wide variety of foot deformities.[64]

Figure 18-13. Custom-molded high top boot designed to fit a patient with Charcot deformity.

There are several randomized clinical trials and prospective cohort studies that describe the benefit of various types of shoes and insoles for high-risk patients with diabetes.[61–64] Most studies include patients with a previous foot ulcer and use a control group of patients with self-selected footwear. In some studies, this is because they cannot afford therapeutic shoes,[62] they refuse recommended shoes,[8] or their insurance does not pay for shoes and insoles.[63] Reulceration is much higher among patients who do not use therapeutic shoes and insoles. About 60% of patients reulcerate with self-selected shoes. Among patients who receive therapeutic shoes and insoles, there is a two- to fourfold reduction in reulceration compared to patients that use shoes they have selected. Even with standard preventative care such as

therapeutic shoes and insoles, education, and regular foot care, about 24% to 50% of patients will develop another foot ulcer within the next year.[61,65]

Reiber et al.[66] conducted a randomized clinical trial and compared self-selected shoes (n = 160), customized medium-density cork inserts with a neoprene closed-cell cover (n = 121), and prefabricated, tapered polyurethane insoles (n = 119). Over a 2-year period, they found no difference in ulcerations in people that had self-selected shoes (17%), cork insoles (15%), and prefabricated insoles (14%). Bus and colleagues[64] evaluated 171 patients with a history of neuropathic ulcerations that received custom footwear that was modified and improved after evaluation in the gait lab compared to patients that receive traditional custom insoles and shoes. Overall, 39% of patients with the custom and improved shoes and insoles reulcerated, and 44% of subjects with traditional custom footwear reulcerated over an 18-month period (P = 0.48). However, when compliance was considered, patients with the custom and modified shoes and insoles had fewer ulcer events (26%) than traditional custom shoes 48% (P = 0.045).

There is a growing body of work that supports the benefit of therapeutic shoes and insoles compared to patient-selected shoes and various applications of custom-made shoes and insoles compared to traditional therapeutic shoes and insoles. Uccioli et al.[67] compared custom-made shoes and insoles to patient-selected shoes and reported a 50% reduction in reulceration with custom-made shoes. Lavery et al.[61] randomized patients to receive a shear-reducing insole compared to a standard insole and off-the-shelf shoe, as well as education and regular foot care, and reported a reduction in reulceration compared to standard-of-care insoles. Ulbrecht and colleagues[68] evaluated 130 patients with a history of foot ulcer that received computer-generated shape- and pressure-based insoles (experimental, n = 66) or standard-of-care insoles (control, n = 64) for 15 weeks. There were significantly fewer ulcers in the group that received custom insoles (9% vs. 25%). Rizzo et al.[69] reported the results of a large cohort study that assessed high-risk patients that received standard care or care within a structured prevention program

with custom shoes and insoles. The cumulative incidence of ulcer in the comprehensive prevention program compared with standard care was 17.6% versus 61% ($P < 0.0001$) after 3 years and 23.5% versus 72% ($P < 0.0001$) after 5 years.

Preventive Education

Education has long been assumed to be an essential component of any program designed to reduce the incidence of DFUs. Preventive education usually takes the form of an intensive introduction to the disease and includes practical steps to cope with the manifestations of diabetes over time. In a 2004 *Cochrane Review*[70] of nine randomized, controlled trials to determine the effectiveness of educational programs in preventing diabetic foot ulceration, the authors concluded that there was only weak evidence to suggest that education reduces foot ulceration and amputations in high-risk patients. On the other hand, the studies reinforced the idea that increased knowledge of foot care had a positive effect on patient behavior in the short term. Continual education and reinforcement may be helpful, especially among high-risk patients, to improve patient outcomes over the long term.

To complicate matters, many patients with diabetes have severe limitations to classical education methods. A large proportion of patients both with and without foot ulcers lacked the visual acuity, manual dexterity, or joint flexibility to perform the self-examination necessary to care for their feet.[5,18] Forty-nine percent of patients with ulcers could not position or see their feet, and 15% were legally blind in at least one eye. When patients are obese or have limited joint mobility or impaired vision, education and self-assessment skills should be directed to both the patient and his or her spouse or caregiver.[5,18] Repetition and regular reinforcement should be practiced by every member of the healthcare team to help the patient and family maintain an understanding of the disease process and continue to practice protective behaviors to avoid some of the serious complications of diabetes.

Temperature Monitoring

At-home temperature monitoring (Fig. 18-14) is a new concept for high-risk patients to identify early warning signs of tissue injury before a foot ulcer actually develops. Because neuropathy inhibits the natural warning system, local inflammation and pain as a result of tissue trauma go unnoticed. Several studies have used temperature assessment as a surrogate to identify tissue injury in patients at risk for DFUs and pressure ulcers. Indeed, two randomized clinical trials demonstrated a 3- to 10-fold reduction in foot complications among high-risk patients using at-home temperature assessment as compared with standard prevention therapy.[71,72]

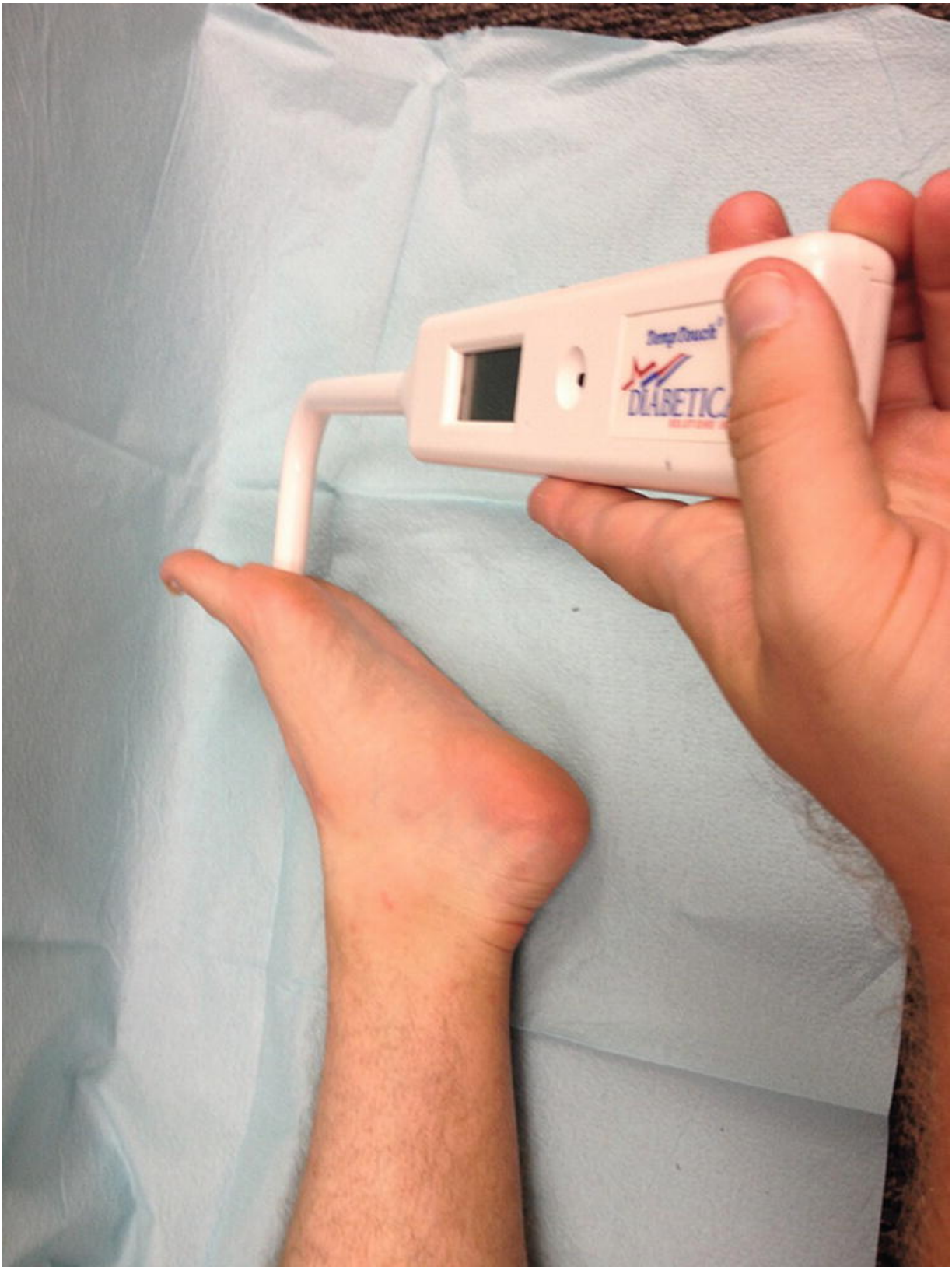

DIABETIC

Figure 18-14. Plantar foot temperature exam. (Photo courtesy of S. van Asten and J. Lafontaine, MD; used with permission.)

Practice Point

At-home temperature monitoring can provide objective feedback to warn patients with neuropathy that their feet are injured before an ulcer develops.

Wound Characteristics and Assessment

Several classification systems can be used to classify diabetic ulcers. The University of Texas ulcer classification system (Table 18-5) is a validated system that includes a mechanism to document wound depth as well as the presence of infection and vascular impairment—two pivotal factors in predicting clinical outcomes. Using this system, the risk of amputation has been shown to be predictive of amputation as wounds increase in depth (grade 0 to III) and progress from no infection (class A), to infection (class B), to PAD (class C), and to infection and PAD (class D) (Fig. 18-15).

Table 18-5 University of Texas Diabetic Wound Classification System

	Grade			
Class	**0**	**I**	**II**	**III**
A	Preulcerative or postulcerative lesion, completely epithelialized	Superficial wound, not involving tendon, capsule, or bone	Wound penetrating to tendon or capsule	Wound penetrating to bone or joint
B	Preulcerative or postulcerative lesion, completely epithelialized with infection	Superficial wound, not involving tendon, capsule, or bone, with infection	Wound penetrating to tendon or capsule with infection	Wound penetrating to bone or joint with infection
C	Preulcerative or postulcerative lesion, completely epithelialized with ischemia	Superficial wound, not involving tendon, capsule, or bone, with ischemia	Wound penetrating to tendon or capsule with ischemia	Wound penetrating to bone or joint with ischemia
D	Preulcerative or postulcerative lesion, completely epithelialized with infection and ischemia	Superficial wound, not involving tendon, capsule, or bone, with infection and ischemia	Wound penetrating to tendon or capsule with infection and ischemia	Wound penetrating to bone or joint with infection and ischemia

Reprinted from Armstrong, D.G., et al. Validation of a Diabetic Wound Classification System: The Contribution of Depth, Infection, and Ischemia to Risk of Amputation. *Diabetes Care* 21(5):855-69, May 1998, with permission.

Figure 18-15. UT1A full-thickness ulcer without involvement of deep structures with no PAD or infection. (Photo courtesy of S. van Asten and J. Lafontaine, MD; used with permission.)

A classification scheme first described by Meggitt and popularized by Wagner has also been used extensively but has the disadvantage of not consistently including wound depth or the presence of infection (Table 18-6). Osteomyelitis is the only type of infection included, and end-stage disease events of gangrene are the only vascular parameters included. Furthermore, the system is difficult to use for more subtle disease processes that are critical for clinical decision-making.

Table 18-6 Meggitt-Wagner Ulcer Classification

Grade	Wound Characteristics
0	Preulceration lesions, healed ulcers, presence of bony deformity
1	Superficial ulcer without subcutaneous tissue involvement
2	Penetration through the subcutaneous tissue; may expose bone, tendon, ligament, or joint capsule
3	Osteitis, abscess, or osteomyelitis
4	Gangrene of digit
5	Gangrene of foot

Reprinted from Wagner, F.W. The Dysvascular Foot: A System for Diagnosis and Treatment. *Foot & Ankle* 2(2):64-122, September 1981; Meggitt, B. Surgical Management of the Diabetic Foot. *British Journal of Hospital Medicine* 16:227-32, 1976, with permission.

The ADA Consensus report[73] recommends that a systematic wound assessment include the following questions in the evaluation:

- Has the patient experienced trauma? Is the ulcer a result of penetrating trauma, blunt trauma, or burn?
- What is the duration of the wound? Is the ulcer acute or chronic?
- What is the progression of local or systemic signs and symptoms? Is the wound getting better, is it stable, or is it deteriorating?
- Has the patient had any prior treatment of the wound or previous wounds? What treatments worked? What failed?

In addition, blood glucose control and comorbidities should be evaluated. Clinical assessment should identify:

- signs of ischemia—adequate blood flow to heal the wound
- signs of soft tissue or bone infection—cellulitis, abscess, or osteomyelitis
- wound depth—undermining or exposed tendon, joint capsule, or bone
- appearance—surrounding callus, devitalized tissue, granulation tissue, drainage, eschar, or necrosis.

Practice Point

Six essentials of the ADA treatment algorithm:

- debridement, early and often
- reducing pressure
- moist wound healing
- treating infection

- correcting ischemia (below the knee disease)
- preventing amputation.

Treatment Plan

Debridement (Surgical, Enzymatic, Maggot, Ultrasound)

Sharp debridement of the ulcer removes devitalized tissue, reduces the bacterial load of the wound, eliminates proteases from the wound bed, and provides a bleeding wound bed. A diabetic ulcer typically has a thick rim of keratinized tissue surrounding it. Debridement must remove all of the callus and devitalized tissue, so that a clean wound edge is created and all edge pressure from the callus is removed. Enzymatic or autolytic debridement may be an option if sharp debridement is not possible or if the patient has PAD.[33] Ongoing debridement may be needed throughout the healing process.[74] Indeed, higher healing rates have been observed in patients who have had more frequent debridement. In addition, in a posthoc evaluation from the becaplermin gel pivotal trial, Steed reported a higher proportion of healed wounds in both the treatment and placebo study groups when wound debridement was performed more frequently.[75]

It is important to acknowledge that not all forms of sharp debridement are the same. Sharp debridement performed in the clinic has significant limitations that may result in insufficient removal of devitalized tissue and pathogens (Fig. 18-16A and B). The inability to control significant bleeding, pain control, and the nonsterility of the environment in which it is performed limit aggressive debridement. Operating room debridement eliminates these barriers. An evolution in thought for the treatment of chronic wounds should include “excisional debridement.”76 This technique describes the excision of the entirety of the wound and its perimeter much like the approach to a soft tissue tumor. Further, in the operating room setting, after this type of debridement is performed, a definitive closure or graft can be placed.

A

Figure 18-16. (A) Full-thickness ulcer with undermining predebridement. **(B)** Ulcer postdebridement (Note: increased in actual size). (Photos courtesy of S. van Asten and J. Lafontaine, MD; used with permission.)

Maggot therapy consists of the topical application of medical-grade, sterile, growth-arrested fly larvae. This type of debridement is reserved for the debridement of nonviable tissue in patients that may not be a candidate for surgical debridement.[77] Sherman reported a significant reduction in necrotic tissue with maggot therapy as compared with conventional wound therapy with complete removal of all necrotic tissue after 4 weeks.[78] Maggots are applied onto the wound surface and are housed in a breathable mesh cage about the wound site for 2

to 3 days at a time. The maggots digest and degrade infected and necrotic tissue continuously without effecting healthy tissue.

Off-loading

Reduction of pressure and shear forces on the foot may be the single most important yet most often neglected aspect of neuropathic ulcer treatment. Off-loading therapy is a key part of the treatment plan for DFUs. The goal is to reduce the pressure at the ulcer site and keep the patient ambulatory.[79,80] Several methods are available to protect the foot from abnormal pressures. Off-loading strategies must be tailored to the age, strength, activity, and home environment of the patient. In general, however, more restrictive off-loading approaches will result in less activity and better wound healing. Education is critical to improve compliance with off-loading. The patient must understand that the wound is a result of repetitive pressure and that every unprotected step is literally tearing the wound apart.

Practice Point

Methods to off-load the diabetic foot include:

- bed rest
- wheelchair
- ambulatory aids (crutches, walker)
- felted foam padding
- half-shoes
- therapeutic shoes
- custom shoes
- custom total contact foot orthoses
- custom splints or braces
- prefabricated cast walkers.

Total Contact Cast

The use of a total contact cast (TCC) is considered the gold standard for off-loading the foot. TCCs reduce pressure at the ulcer site while still allowing the patient to be ambulatory.[81] A skilled clinician or technician is required to apply the molded plaster cast to ensure a proper fit. A TCC is a modification of a traditional fracture cast that uses minimal cast padding and includes a covering to protect the toes (Fig. 18-17A–D). The cast is molded to the contour of the foot and leg so that no movement is possible within the cast. TCCs are generally changed every 1 to 2 weeks but may need to be replaced more frequently in patients with edema or other concerns.

B

C

Figure 18-17. Applying a total contact cast (TCC). **(A)** A foam layer covers the toes for protection, and padding is applied over bony prominences before the first layer of casting material is applied. **(B)** Application of the TCC. **(C)** Completed TCC. **(D)** A cast boot covers the TCC. (Photo courtesy of J. McGuire, DPM. Used with permission.)

A TCC is one of the most effective ways of treating plantar neuropathic foot ulcers.[81–85] Numerous studies[82,85] have shown that TCCs can heal ulcers in 6 to 8 weeks. In descriptive and randomized clinical trials, the proportion of wounds that heal with TCCs is consistently much higher than those using topical growth factors, bioengineered tissue, or special dressings.[86,87]

One of the main advantages of using a TCC is that it encourages patient compliance with off-loading. The ulcer is protected with every step the patient takes. Using a TCC to facilitate wound healing is analogous to using a cast to heal a fracture—in both cases, healing is facilitated by rest and immobilization. The TCC reduces the patient's activity level, decreases stride length and cadence, and significantly reduces pressure at the ulcer site.[81,83] The main disadvantages for patients are the same as their complaints with a fracture cast—a cast is heavy and hot and makes bathing, walking, and sleeping difficult.

Practice Point

TCCs *should not* be used if wound infection is suspected or present.

Removable Cast Walkers

The effectiveness of removable cast walkers (Fig. 18-18A–C) to reduce pressure at ulcer sites has been shown in several studies to be comparable to that of TCCs.[81,83] Many practitioners consider removable cast walkers to be their preferred off-loading device because they are less time consuming and easier to apply than TCCs, and they are more readily accepted by patients. In addition, the TCC has several precautions and contraindications that aren't issues with removable walkers. Edema can be overcome with constant adjustments to the fit of the device, and compression dressings can be applied in conjunction with the removable cast boot (Fig. 18-19). Wounds can be inspected regularly and treated with advanced wound care products such as growth factors, electrical stimulation, and other biologically active dressings. Because the wound and limb can be inspected frequently, the vascular concerns inherent in the occlusive irremovable TCC aren't an issue.

A

B

Figure 18-18. (A) Off-loading walker, lateral view. **(B)** Off-loading walker, posterior view. **(C)** Off-loading walker, anterior view with honey comb off-loading insole visible. (Photos courtesy of S. van Asten and J. Lafontaine, MD; used with permission.)

Figure 18-19. Removable cast boot with a removable cushioned foot bed. (Photo courtesy of S. van Asten and J. Lafontaine, MD; used with permission.)

Additional advantages of removable walking boots (as compared with TCCs) are that they are relatively inexpensive, the protective insole can be easily replaced if it shows signs of wear, no special training is required for correct and safe application, and they can be easily removed to assess and debride the wound as appropriate.[88,89] It's also possible to modify removable walkers into nonremovable devices by securing the walker with cast material or a nonremovable cable tie; this is known as an instant TCC. If patients can't remove the walker, the element of compliance that makes the TCC attractive is maintained, and the outcomes for healing improve to the levels seen with the TCC.[90,91]

No one off-loading device is appropriate for every patient. McGuire[92] has suggested a transitional approach to healing and maturing the DFU that uses the instant TCC for initial pressure management and transitions to removable devices and shoe-based platforms before the patient is ready for definitive footwear.

In a randomized controlled trial, Armstrong et al.[93] compared the effectiveness of TCCs, removable cast walkers, and half-shoes in healing neuropathic foot ulcerations in individuals with diabetes. The percentage of healing at 12 weeks was 89.5% for the TCC, 65.0% for the cast walker, and 58.3% for the half-shoe. When the cast walker is made nonremovable (ITCC), the difference between the TCC and cast walker effectively disappears.[94]

Healing Sandals and Half-Shoes

A number of healing sandals and half-shoes or wedged shoes are available to reduce pressure on the forefoot (Fig. 18-20A–C). These sandals and shoes are useful for patients who can't tolerate a TCC or for those who need a transitional device after removal of a TCC while they're awaiting custom-made therapeutic shoes and insoles. A modification of the Carville healing sandal can be made from a standard surgical shoe with a total contact direct-molded Plastazote insole.[95]

B

C

Figure 18-20. (A) Lateral view of healing sandal. **(B)** Rear view of healing sandal. **(C)** Top view with off-loading honey comb insole removal (individual pieces can be removed for additional off-loading). (Photos courtesy of S. van Asten and J. Lafontaine, MD; used with permission.)

Surgical shoes with a rocker sole design are preferable to the flat design for postoperative use. Royce Medical has a healing sandal that utilizes the Active Hex insole, described above, and that can be used as a transitional device after closing the wound.

The Wedge shoe (Fig. 18-21) by Darco products was originally designed to protect the forefoot after elective surgery. This shoe has a sole that's wedged at a 10-degree dorsiflexion angle, effectively removing pressure from the forefoot area. A study by Lair[96] provides support for its role in postoperative patients following surgery on the forefoot. However, these types of shoes aren't well accepted by patients because they're difficult to walk in, they typically cause pain of the contralateral extremity, and they are not safe for use in patients with postural instability. Also, many people with diabetes have equinus and cannot tolerate the negative heel position created by the shoe. Further, suspension of the heel during ambulation increases pressure on the forefoot and stresses the midfoot, a common site for collapse in the diabetic Charcot foot. In a randomized clinical trial that compared TCCs with healing sandals and removable cast boots, patients in the healing sandal group were less compliant and used the device during walking significantly less than did subjects in the TCC group.[97]

Figure 18-21. Wedged forefront-relief shoe.

Ankle–Foot Orthoses

Custom-made ankle–foot orthoses can be used for lower extremity pathology, including Charcot fractures, tendon injuries, and neuropathic ulcers. The Charcot restraint orthotic walker (Fig. 18-22), for example, initially was used to treat patients with neuropathic fractures. It provides protection to the neuropathic foot and aids in controlling lower extremity edema. This device looks like a ski boot; it has a rigid polypropylene shell with a rocker bottom sole.

Figure 18-22. Charcot restraint orthotic walker.

The primary drawback to custom-made devices is that they typically cost more than $1,000. If the structure of the foot changes or local

edema resolves, the device can no longer be used. Since a number of less expensive, off-the-shelf products are now available to treat neuropathic wounds, custom ankle–foot orthoses are used less commonly. Off-the-shelf devices should be replaced at regular intervals because the materials in the insoles will lose their effectiveness over time.

Negative Pressure Wound Therapy

Negative pressure wound therapy (NPWT) is widely recognized as an effective adjunctive therapy in diabetic foot wound healing and has been reported to increase the rate of healing and promote the formation of granulation tissue[98] (Fig. 18-23). NPWT utilizes the application of subatmospheric pressure inducing micro tissue deformation that stimulates the healing cascade.[99] Although NPWT can be used for extended periods of time in both the inpatient and outpatient settings, there should be a clear goal of therapy in mind prior to its implementation. NPWT can be used to terminal epithelialization in some cases; however, in the opinion of the authors, NPWT should be principally used as an adjunctive wound bed preparation tool that decreases wound depth and provides a granular wound base for graft (autograft, allograft, or xenograft) acceptance.

Figure 18-23. Negative pressure wound therapy, inpatient setting. (Photo courtesy of S. van Asten and

J. Lafontaine, MD; used with permission.)

The use of NPWT devices provides greater portability with more reliable mechanisms and potentially most impactful because simultaneous irrigation can be combined with the therapy. There are two approaches that are used. One approach is to provide continuous irrigation of solution with NPWT. The second approach is to “instill” fluid into the sponge without NPWT for a specified period of time, so the wound is soaked with irrigation solution. The NPWT is continued, and the solution is removed from the field. Negative pressure wound therapy with instillation (NPWTi) (Fig. 18-24) allows for preprogrammed cycles of installation of a solution and the application of negative pressure. In the setting of infection, NPWTi devices have been reported to have a significant impact on a variety of wounds. Lehner and colleagues reported greater than 80% salvage rate for acute and chronically infected total joint implants utilizing NPWTi,[100] and Kim and colleagues reported a significant reduction in the number of surgeries and length of hospital stay for patients with acutely infected wound requiring hospitalization.[90,101] The ideal type of solution, dwell time, duration of therapy, and other questions are unclear.[102]

Figure 18-24. Next-generation negative pressure wound therapy.

Electrical Stimulation

Several studies have suggested a beneficial effect of electrical stimulation on the acceleration of wound healing, increase of cutaneous perfusion, and cellular immunity.[103] Although there is a substantial body of work that supports the effectiveness of electrical stimulation in wound healing, most surgeons have a poor understanding of the technology and its potential applications in patients with diabetes. All living human cells are enveloped by a plasma membrane that operates on the electrochemical physiology of direct current exchange of ions.[104] Injury to the epithelial layer of the human skin disrupts the natural current of these ions, creating an electrical field and changing the movement of epithelial cells and fibroblasts.[105] This cellular migration may be reversed with the use of electrical stimulation by changing the polarity of the electrical field back in its natural electric current.[106]

The application of transcutaneous electrical nerve stimulation (TENS) to improve skin perfusion and venous flow in diabetic patients is reported frequently.[107] When the human skin becomes hypoxic and releases cytokines, the expression of vascular endothelial growth factor (VEGF) is increased. Electrical stimulation initiates the release of stored VEGF followed by an increase in gene expression of VEGF. Daily electrical stimulation might allow secretion of VEGF to remain above basal levels throughout the healing process.

Bacterial load and infection seem to be important factors in chronic wounds and delayed healing of leg ulcers of people with diabetes.[108] The bacteriostatic effect of electrical stimulation is described in several experimental studies[109] and may lower the bioburden in the wound bed. Future clinical trials are needed to fully understand the influence of electrical stimulation on infected leg ulcers in diabetics.

Other Adjunctive Therapies

The suggestion that hyperbaric oxygen therapy (HBO) is applicable to lower extremity pathology has been a point of discussion for many years among healthcare professionals. Most reviews of medical publications about HBO treatment and the diabetic foot report little evidence to support the choice of oxygen therapy as an adjunctive therapy for wound healing.[110] However, a high-quality trial has demonstrated a beneficial effect of HBO on the healing of DFUs.[111] Although a few limitations can be listed about the study, additional randomized control trials need to be repeated to investigate its further implications. Given the high economic burden of this treatment modality, studies with full health economic analysis are needed before HBO sessions can be routinely incorporated into clinical practice.

Many products have been designed to correct aspects of the wound biochemistry and the cell biology of diabetic ulcers. Some data suggest the use of endothelial growth factor (EGF) to hasten healing.[112] Dressings, skin substitutes, and even platelet-derived products are being studied to enhance the healing of chronic ulcers in the diabetic foot.[113]

Antibiotics

Without appropriate wound care, antibiotic therapy is often insufficient to treat infected ulcers. Only clinically infected wounds require an empirical antibiotic regimen, based on the severity of the infection and the likely etiologic agent(s). Mild-to-moderate infections in patients who have not recently received antibiotic therapy may be sufficiently treated with a therapy aimed solely at aerobic gram-positive cocci (*Staphylococcus aureus, β-hemolytic streptococci*). Severe infections or recent antibiotic therapy require broad-spectrum empirical therapy, to overcome resistant organisms.[42]

Because of limited evidence with which to make choices among antibiotic regimens, the route of therapy, and the duration of antibiotic therapy, no single combination appears to be superior to others.[114] In case of osteomyelitis, generally, a duration of at least 4 to 6 weeks is required, and probably a longer duration is needed if infected bone remains.

Topical Antiseptics and Wound Cleansers

Although antiseptics and wound cleansers are ubiquitously utilized, their importance to wound care has been underappreciated. Their value lies in their ability to remove devitalized tissue as well as reduce bioburden. Unlike antibiotic therapy, antiseptics are less likely to develop resistance to microbes, a concentrated localized effect, are generally better tolerated, and are widely available. Commonly used antiseptics include Dakin's solution (dilute bleach), dilute acetic acid, polyhexanide, and dilute iodine. In vitro studies have repeatedly demonstrated significant efficacy against a wide range of bacteria, yeast, and fungus including resistant strains.[115,116] In light of the troubling trend of growing antimicrobial resistance, topical antiseptics and wound cleansers may provide an important treatment modality.

Antiseptic therapy can be applied in different ways. With every dressing change, the wound can be washed with an antiseptic solution. It can also be applied onto the wound surface multiple times daily via an antiseptic saturated contact dressing (e.g., 4 × 4 dressing). Continuous or intermittent delivery via NPWT devices can also be used in the acute setting to instill antiseptic solutions.

Along the same category, another widely available yet generally underutilized topical therapy in wound care are surfactants. Surfactants are detergents that break up debris and prevent adherence of foreign materials. Surfactants have been utilized for orthopedic application for many years in the removal of acute contamination as well as biofilm reduction on hardware.[117,118] For example, castile soap is well tolerated and widely available and has been reported to be effective. Anglen reported that castile soap worked as well as bacitracin solution with less wound healing problems in 458 open lower extremity open fractures.[119]

Surgery to Heal Wounds and Prevent Recurrence

Is there good evidence that foot surgery in patients who have diabetes will prevent ulceration in the future? The question for physicians and patients is whether the risks of surgery are better than the risk of having a chronic foot ulcer. The risks of infection and amputation from a nonhealing foot ulcer are high. Approximately 10% to 20% of DFUs end in amputation,[120,121] 56% are treated for infection, and 20% develop osteomyelitis.[7,18,122] Ulcer recurrence, as previously discussed, is about 30% per year when standard preventative therapies are provided. The incidence of ulceration is 50% to 80% when no additional prevention is provided. On the other hand, several authors have reported the results of planned surgical procedures to heal foot ulcers. These studies suggest a high rate of wound healing (91% to 100%) and a low rate of ulcer recurrence after 2 years (0% to 39%).[123–125] If surgery is simply viewed as a prevention tool, in the correct subpopulation, surgery has the lowest reulceration rate. The goal of surgery is to reduce the long-term risk for reulceration by increasing joint motion where it is limited, reducing abnormal pressure points, and repairing structural foot deformities when they are an underlying cause of ulceration. The literature has several reports of retrospective case series, but there is only one randomized clinical trial that reports clinical outcomes with this type of approach. In general, surgery seems to be safe and effective at healing recalcitrant ulcers and reducing the risk of reulceration.

There is no evidence that elective surgery such as hallux valgus or hammer toe correction reduces the risk of future ulceration in patients with no history of ulceration. Patients with diabetes should undergo elective foot surgery only if they have severe deformity, pain, or functional limitations that warrant surgery rather than an expectation that surgery will prevent a foot ulcer in the future. However, there is evidence that these types of surgeries are effective to heal foot ulcers and reduce the risk of reulceration. For example, percutaneous lengthening of the Achilles tendon[126] has been shown to reduce plantar foot pressures in subjects with prior ulceration (Fig. 18-25).

The rationale for surgery is that limited active ankle joint range of motion causes more pressure and shear stresses on the ball of the foot, leading to ulceration. Mueller and colleagues[127] demonstrated a reduction of about 27% reduction in forefoot loading after lengthening the Achilles as well as a reduced risk of reulceration. In a randomized clinical study, Mueller[127] compared Achilles tendon lengthening to TCCs in 61 patients with DFUs. The incidence of healing was the same in both groups (Achilles surgery 100% TCC 88%); however, the incidence of reulceration over 2 years was 38% and 81%, respectively. Lin and colleagues[128] reported on a cohort of patients with DFUs that failed to heal after a period of immobilization via a TCC. Using the Achilles lengthening procedure, 93% of patients (14/15) healed in an average of 39 days with no ulcer recurrence in the subsequent 17 months.

Figure 18-25. Percutaneous Achilles tendon lengthening can assist in forefoot ulcer healing or prevent recurrence by decreasing peak plantar

pressure. (Photo courtesy of S. van Asten and J. Lafontaine, MD; used with permission.)

Another example of selective foot surgery to reduce the risk of ulcer recurrence involves ulcers of the great toe. Foot ulcers of the great toe are commonly associated with hallux rigidus or a reduced range of motion of the first metatarsophalangeal joint.[129,130] One of the most common surgical approaches to address hallux rigidus in patients with diabetes is a resectional arthroplasty of the first metatarsophalangeal joint or more specifically resection of a portion of the base of the proximal phalanx of the great toe (Fig. 18-26). This is also referred to as a Keller bunionectomy. The Keller resectional arthroplasty removes the arthritic joint and allows a pseudoarthrosis to develop in its place. Like the surgery described to increase ankle joint range of motion by lengthening the Achilles tendon, arthroplasty of the great toe has been reported to increase healing of ulcers that have failed other therapies with a much lower rate of ulcer recurrence. Armstrong reported the results of a cohort study of 41 diabetic patients with great toe ulcers.[125] Patients either received resectional arthroplasty of the great toe or standard wound care. The surgery group had faster healing (24 vs. 67 days) and few recurrent ulcers after the surgery (5% vs. 35%).

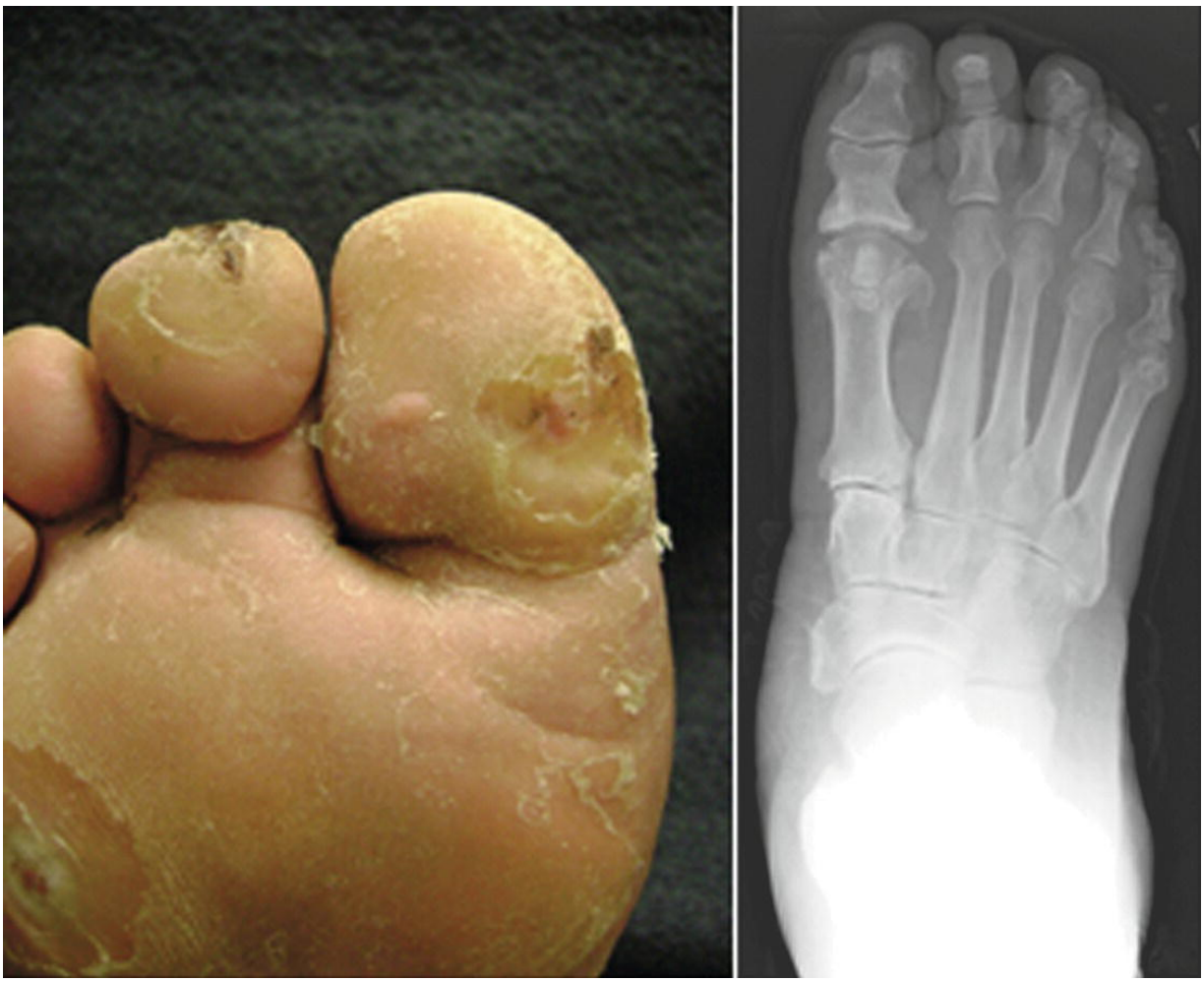


Figure 18-26. Plantar ulceration, healed after resection of the base of the proximal phalanx of the hallux (Keller arthroplasty). Notice the shortened proximal phalanx of the hallux. (Photos courtesy of S. van Asten and J. Lafontaine, MD; used with permission.)

Empowering and Engaging Patients with DM

Diabetes is a chronic complex disease that requires the patient to perform self-assessments of blood glucose levels, administer medications, and make daily self-management decisions. The therapeutic relationship between the patient with DM and the healthcare professional should be one that empowers the patient by using appropriate language that engages and motivates the patient. The importance of how to speak with patients with diabetes is an important part of the care and treatment (American Association of Diabetes Educators, American Diabetes Association, Dickerson, Guzman, Maryniuk, 2017a, Dickerson, Guzman, Maryniuk, et al. 2017b, Dickinson & Funnell, 2019, Funnell, 2016). (See Table 18-7 for some suggestions from Dickinson and Funnell (2019) about empowering message that health care professionals should consider when speaking with patients with diabetes.)[131–136]

Table 18-7 Empowering Messages[a,b]

Terms to Avoid	Suggested Alternatives
Compliant and noncompliant; compliance and noncompliance; adherent and nonadherent; adherence and nonadherence	"He takes his medication about half the time"; "she takes insulin whenever she can afford it"; "he eats fruits and veggies a few times a week"; engagement; participation; involvement; medication taking
Control (as a verb or an adjective): controlled and uncontrolled; well-controlled and poorly controlled	"She is checking her blood glucose levels a few times a week"; "he is taking sulfonylureas, and they are not bringing his blood glucose levels down enough"; manage
Control (as a noun): glycemic control; glucose control; poor control; good control; bad control; tight control	Hemoglobin A1C; blood glucose levels; blood glucose targets; glycemic target or goal; glycemic stability; glycemic variability
Diabetic (as an adjective): diabetic person ("How long have you been diabetic?"); diabetic foot; diabetic education; diabetes patients	Person with diabetes ("How long have you had diabetes?"); foot ulcer or infection on the foot; diabetes education; patients with diabetes
Diabetic (as a noun): nondiabetic; normal; "Are you a diabetic?"	Person living with diabetes; person with diabetes; person who has diabetes; person who does not have diabetes; person without diabetes; "Do you have diabetes?"
Imperatives: can and cannot; should and should not; do and do not; have to and need to; must and must not	"Have you tried…"; "what about…"; "may I suggest…"; "may I tell you what has worked for other people…"; "what is your plan for…"; "would you like to consider…"
Regimen; rules	Plan; choices
Words or phrases that focus on the provider: "I got him/her to…"; "I want you to…"; "Let people…"; "Setting goals for…"	"He started taking insulin…"; "she lost 25 pounds…"; "may we make a plan for…"; "may I suggest…"; facilitating identified goals and creating a plan with; self-directed goals
Refused	Declined
Victim; suffer; stricken; afflicted	Lives with diabetes; has diabetes; diagnosed with diabetes
Words or phrases that imply judgment: lifestyle disease; difficult patient; in denial	"Ms. Smith has a foot sore that is not healing and is having a difficult time with off-loading"; "I'm having a difficult time with Ms. Smith"; "Dan understands that diabetes can harm him, but he does not see diabetes as a priority with everything else that's going on in his life right now."
Unmotivated; unwilling	"John has not started taking insulin because he's concerned about weight gain. He sees insulin as a personal failure."
"What did you do?"	"Tell me about…"; "may I suggest?"
Cheating; sneaking	Making choices and decisions
Good; bad; poor	Numbers; choices; food; safe or unsafe
Fail; failed; failure; "She failed metformin."	"Metformin was not adequate to reach her hemoglobin A1C goal."
Test; test blood glucose; test strips	Check blood glucose; blood glucose monitoring; strips, glucose strips
Words or phrases that threaten "You are going to end up blind or on dialysis."	"More and more people are living long and healthy lives with diabetes. Let's work together to make a plan that works for you."

[a]Adapted with permission from the American Diabetes Association: Dickinson JK, Guzman SJ, Maryniuk MD, et al. The use of language in diabetes care and

education. *Diabetes Care*. 2017;40(12):1790–1799.[b]Adapted with permission from SAGE Publishing: Dickinson JK, Guzman SJ, Maryniuk MD, et al. The use of language in diabetes care and education. *Diabetes Educ*. 2017;43(6):551–564.Reprinted with permission from Dickinson, J.K., Funnell, M.M. Diabetes: Changing the Conversation. *Nursing* 49(6):57-60, 2019.

Summary

DFU care is a challenge for both the patient and the healthcare provider. As the population continues to age, the incidence of diabetes will continue to increase, which will in turn lead to more diabetic wounds. A team approach—with total involvement of the healthcare system and the necessary partnership with the patient—will be the infrastructure for achieving better outcomes of care.

Early assessment for the risk factors for foot ulceration in patients with diabetes is essential. A variety of methods must be used to identify at-risk patients. Appropriate skin care and properly fitting shoes are mandatory for any patient with diabetes. Clinicians can choose among many products available to off-load the diabetic foot and to treat the diabetic foot. Infection is an important concern in diabetic ulcers and warrants prompt identification and treatment. Adjuvant therapies coupled with debridement and appropriate dressings can be critical in salvaging the diabetic limb. Future research is needed to determine appropriate therapy combinations and individual treatment plans for patients with DFUs (Box 18-1).

PATIENT SCENARIO

A 51-year-old Caucasian male with diabetes and a neuropathic ulcer to the plantar aspect of his right foot arrives at the hospital (Fig. 18-27A). He was already treated for his foot ulcer in a local hospital wound care center with periodic debridement and HBO. He has a history of peripheral neuropathy and a high level of activity as he manages a ranch.

His past medical history includes control visits for his diabetes (12 years), hypertension, hyperlipidemia, and coronary artery disease. The wound on his foot has been managed with standard of care including off-loading, debridement, enzymatic debridement, and moist wound care. His current medications include metformin, glipizide, enalapril, and Toprol XL. He has a significant history of 40 pack-years

of smoking but quit 19 years ago. He denies any alcohol consumption.

His physical examination reveals an obese man, very pleasant and oriented. He demonstrates barely palpable dorsalis pedis pulse and barely palpable posterior tibial pulse in both feet. The capillary filling time is less than 3 seconds. He demonstrates LOPS to 10/10 sites in both feet. He has absent pedal hair and dry, xerotic skin. The patient is unable to feel the Semmes-Weinstein monofilament at any site tested, revealing significant bilateral neuropathy. He has no pain or temperature sensation in either foot. His right foot has an ulcer under the head of the first metatarsal; the base of the ulcer is red with surrounding hyperkeratotic tissue, undermining the skin margins. The ulcer measures 1.9 × 1.2 × 0.3 cm after debridement (Fig. 18-27B). No bone can be palpated when the wound is examined with a sterile probe. Biomechanical evaluation reveals a plantar-flexed, non–weight-bearing first metatarsal with significant equinus bilaterally. Laboratory evaluation is within normal limits. Radiographs reveal no foreign body, nor signs of osteomyelitis.

The wound is assigned a grade of 1A on the University of Texas classification system. There are concerns about his blood flow status. Noninvasive studies are ordered and reveal a slightly decreased left ABI of 0.91 and biphasic waveforms at the anterior tibial artery and the posterior tibial artery. Transcutaneous oximetry measurements are performed and demonstrate an increase (33 to 54 mm Hg) at the midfoot level. The vascular surgeon is consulted and recommends that the ulcer has to heal without curative surgery.

The wound is stagnant for a period of 2 months. Off-loading devices attempted include removable CAM boot and a half (wedge) shoe. Weekly to biweekly debridement with TCC allows the wound to heal in 6 weeks (Fig. 18-27C).

A

B

Figure 18-27. (A) Ulcer appearance at the time of presentation. **(B)** Ulcer appearance after debridement. **(C)** Complete epithelialization of ulcer at 6 weeks. (Photos courtesy of S. van Asten and J. Lafontaine, MD; used with permission.)

Show What You Know

1. **According to the ADA, how many people are unaware that they have diabetes mellitus?**
   A. 50%
   B. 30%
   C. 75%
   D. 25%

2. **The single leading cause of lower extremity amputation is:**
   A. diabetes mellitus.
   B. lymphedema.
   C. arterial occlusion.
   D. venous disease.

3. **According to the ADA, good skin care includes all of the following *except*:**
   A. keeping skin clean and dry.
   B. applying moisturizers between toes.
   C. avoiding very hot showers and tub baths.
   D. checking feet daily for cracks or fissures.

4. **Off-loading strategies must be tailored to the age, strength, activity, and home environment of the patient.**
   A. True
   B. False

5. **For which of the following treatment strategies have healing rates for diabetic foot ulcers been found to be comparable to those with total contact casts (TCC)?**
   A. Wedged shoe
   B. Half-shoe
   C. Wide-toe shoe
   D. Walker boot rendered irremovable (ITCC)

Answers to these questions are found on page 686.

References

1. Ogurtsova, K., et al. Idf Diabetes Atlas: Global Estimates for the Prevalence of Diabetes for 2015 and 2040. Diabetes Research and Clinical Practice 128:40-50, 2017.
2. National Health Interview Survey (NHIS, available at http://www.cdc.gov/nchs/nhis.htm) of the National Center for Health Statistics (NCHS), Centers for Disease Control and Prevention (CDC). United States, April 2017.
3. American Diabetes Association. Economic Costs of Diabetes in the U.S. in 2017. Diabetes Care 41(5): 917-28, 2018.
4. Lavery, L.A., Armstrong, D.G., Harkless, L.B. Classification of Diabetic Foot Wounds. Journal of Foot and Ankle Surgery 35(6):528-31, 1996.
5. Lavery, L.A., et al. Practical Criteria for Screening Patients at High Risk for Diabetic Foot Ulceration. Archives of Internal Medicine 158(2):157-62, 1998.
6. Lavery, L.A., et al. Amputations and Foot-Related Hospitalisations Disproportionately Affect Dialysis Patients. International Wound Journal 12(5):523-6, 2015.
7. Lavery, L.A., et al. Reevaluating the Way We Classify the Diabetic Foot: Restructuring the Diabetic Foot Risk Classification System of the International Working Group on the Diabetic Foot. Diabetes Care 31(1):154-6, 2008.
8. Edmonds, M.E., et al. Improved Survival of the Diabetic Foot: The Role of a Specialized Foot Clinic. Quarterly Journal of Medicine 60(232):763-71, 1986.
9. Dargis, V., et al. Benefits of a Multidisciplinary Approach in the Management of Recurrent Diabetic Foot Ulceration in Lithuania: A Prospective Study. Diabetes Care 22(9):1428-31, 1999.
10. Wukich, D.K., et al. Outcomes of Osteomyelitis in Patients Hospitalized with Diabetic Foot Infections. Foot and Ankle International 37(12):1285-1291, 2016.
11. Lavery, L.A., et al. Risk Factors for Foot Infections in Individuals with Diabetes. Diabetes Care 29(6):1288-93, 2006.
12. Lavery, L.A., et al. Validation of the Infectious Diseases Society of America's Diabetic Foot Infection Classification System. Clinical Infectious Diseases 44(4): 562-5, 2007.
13. Centers for Disease Control and Prevention. National Diabetes Statistics Report, 2017. US Department of Health and Human Services, CDC, 2017.
14. Lavery, L.A., et al. Diabetes-Related Lower-Extremity Amputations Disproportionately Affect Blacks and Mexican Americans. Southern Medical Journal 92(6):593-9, 1999.
15. Lavery, L.A., et al. Variation in the Incidence and Proportion of Diabetes-Related Amputations in Minorities. Diabetes Care 19(1):48-52, 1996.
16. Chukwueke, I., Cordero-MacIntyre, Z. Overview of Type 2 Diabetes in Hispanic Americans. International Journal of Body Composition Research

8(Suppl):77-81, 2010.
17. Lavery, L.A., Peters, E.J., Armstrong, D.G. What Are the Most Effective Interventions in Preventing Diabetic Foot Ulcers? International Wound Journal 5(3):425-33, 2008.
18. Armstrong, D.G., et al. Choosing a Practical Screening Instrument to Identify Patients at Risk for Diabetic Foot Ulceration. Archives of Internal Medicine 158(3):289-92, 1998.
19. Calhoun, J.H., et al. Diabetic Foot Ulcers and Infections: Current Concepts. Advances in Wound Care Journal 15(1):31-42; quiz 44-5, 2002.
20. Wunderlich, R.P., et al. Defining Loss of Protective Sensation in the Diabetic Foot. Advances in Wound Care 11(3):123-8, 1998.
21. Persaud, R., Coutts, P.M., Brandon, A., et al. Validation of the Healthy Foot Screen: A Novel Assessment Tool for Common Clinical Abnormalities. Advances in Skin & Wound Care 31(4):154-62, April 2018.
22. Sibbald, R.G., Ayello, E.A., Alavi, A., et al. Screening for the High-Risk Diabetic Foot: A 60-Second Tool (2012). Advances in Skin & Wound Care 25(10):465-76, October 2012.
23. Woodbury, M.G., Sibbald, R.G., Ostrow, B., et al. Tool for Rapid & Easy Identification of High Risk Diabetic Foot: Validation & Clinical Pilot of the Simplified 60 Second Diabetic Foot Screening Tool. PLoS One 10(6):e0125578, 2015.
24. Levin, M. Diabetic Foot Wounds: Pathogenesis and Management. Advances in Wound Care 10(2):24-30, 1997.
25. Perkins, B.A., et al. Simple Screening Tests for Peripheral Neuropathy in the Diabetes Clinic. Diabetes Care 24(2):250-6, 2001.
26. Oyer, D.S., Saxon, D., Shah, A. Quantitative Assessment of Diabetic Peripheral Neuropathy with Use of the Clanging Tuning Fork Test. Endocrine Practice 13(1):5-10, 2007.
27. Lavery, L.A., et al. Accuracy and Durability of Semmes-Weinstein Monofilaments: What Is the Useful Service Life? Diabetes Research and Clinical Practice 97(3):399-404, 2012.
28. Booth, J., Young, M.J. Differences in the Performance of Commercially Available 10-g Monofilaments. Diabetes Care 23(7):984-8, 2000.
29. Yong, R., et al. The Durability of the Semmes-Weinstein 5.07 Monofilament. Journal of Foot and Ankle Surgery 39(1):34-8, 2000.
30. Tooke, J.E., Brash, P.D. Microvascular Aspects of Diabetic Foot Disease. Diabetic Medicine 13(Suppl 1): S26-9, 1996.
31. Chao, C.Y., Cheing, G.L. Microvascular Dysfunction in Diabetic Foot Disease and Ulceration. Diabetes/Metabolism Research and Reviews 25(7):604-14, 2009.
32. LoGerfo, F.W., Misare, B.D. Current Management of the Diabetic Foot. Advances in Surgery 30:417-26, 1996.
33. Mulder, G.D. Evaluating and Managing the Diabetic Foot: An Overview. Advances in Skin & Wound Care 13(1):33-6, 2000.

34. Hirsch, A.T., et al. ACC/AHA Guidelines for the Management of Patients with Peripheral Arterial Disease (Lower Extremity, Renal, Mesenteric, and Abdominal Aortic): A Collaborative Report from the American Associations for Vascular Surgery/Society for Vascular Surgery, Society for Cardiovascular Angiography and Interventions, Society for Vascular Medicine and Biology, Society of Interventional Radiology, and the ACC/AHA Task Force on Practice Guidelines (writing committee to develop guidelines for the management of patients with peripheral arterial disease)—Summary of Recommendations. Journal of Vascular and Interventional Radiology 17(9):1383-97; quiz 1398, 2006.
35. Catanzariti, A.R., et al. Off-Loading Techniques in the Treatment of Diabetic Plantar Neuropathic Foot Ulceration. Advances in Wound Care 12(9):452-8, 1999.
36. Lavery, L., Gazewood, J.D. Assessing the Feet of Patients with Diabetes. Journal of Family Practice 49(11 Suppl):S9-16, 2000.
37. Lavery, L.A., et al. Ankle Equinus Deformity and Its Relationship to High Plantar Pressure in a Large Population with Diabetes Mellitus. Journal of the American Podiatric Medical Association 92(9): 479-82, 2002.
38. Grant, W.P., et al. Electron Microscopic Investigation of the Effects of Diabetes Mellitus on the Achilles Tendon. Journal of Foot and Ankle Surgery 36(4):272-8; discussion 330, 1997.
39. Berendt, A.R., et al. Diabetic Foot Osteomyelitis: A Progress Report on Diagnosis and a Systematic Review of Treatment. Diabetes/Metabolism Research and Reviews 24(Suppl 1):S145-61, 2008.
40. Lipsky, B.A., et al. Diagnosis and Treatment of Diabetic Foot Infections. IDSA Guidelines. Clinical Infectious Diseases 39(7):885-910, 2004.
41. Bergamaschini, L., et al. Complement Activation in Diabetes Mellitus. Journal of Clinical and Laboratory Immunology 35(3):121-7, 1991.
42. Kim, S.H., et al. Effects of Acute Hyperglycemia on Endothelium-Dependent Vasodilation in Patients with Diabetes Mellitus or Impaired Glucose Metabolism. Endothelium 10(2):65-70, 2003.
43. Geerlings, S.E., Hoepelman, A.I. Immune Dysfunction in Patients with Diabetes Mellitus (DM). FEMS Immunology and Medical Microbiology 26(3-4):259-65, 1999.
44. Cavalot, F., et al. Insulin, at Physiological Concentrations. Enhances the Polymorphonuclear Leukocyte Chemotactic Properties. Hormone and Metabolic Research 24(5):225-8, 1992.
45. Chakraborti, C., Le, C., Yanofsky, A. Sensitivity of Superficial Cultures in Lower Extremity Wounds. Journal of Hospital Medicine 5(7):415-20, 2010.
46. Perry, C.R., Pearson, R.L., Miller, G.A. Accuracy of Cultures of Material from Swabbing of the Superficial Aspect of the Wound and Needle Biopsy in the Preoperative Assessment of Osteomyelitis. The Journal of Bone and Joint Surgery. American Volume 73(5): 745-9, 1991.

47. Senneville, E., et al. Culture of Percutaneous Bone Biopsy Specimens for Diagnosis of Diabetic Foot Osteomyelitis: Concordance with Ulcer Swab Cultures. Clinical Infectious Diseases 42(1):57-62, 2006.
48. Wolcott, R.D., et al. Biofilm Maturity Studies Indicate Sharp Debridement Opens a Time-Dependent Therapeutic Window. Journal of Wound Care 19(8): 320-8, 2010.
49. Harrison-Balestra, C., et al. A Wound-Isolated Pseudomonas Aeruginosa Grows a Biofilm In Vitro within 10 Hours and Is Visualized by Light Microscopy. Dermatologic Surgery 29(6):631-5, 2003.
50. Davis, S.C., et al. Microscopic and Physiologic Evidence for Biofilm-Associated Wound Colonization In Vivo. Wound Repair and Regeneration 16(1):23-9, 2008.
51. Costerton, J.W., Stewart, P.S., Greenberg, E.P.. Bacterial Biofilms: A Common Cause of Persistent Infections. Science 284(5418):1318-22, 1999.
52. Fromantin, I., et al. Occurrence and Persistence of Biofilms on Cared Chronic Wounds: A Large Multicentric Clinical Study. Wound Medicine 23:28-34, December 2018.
53. James, G.A., et al. Biofilms in Chronic Wounds. Wound Repair and Regeneration 16(1):37-44, 2008.
54. Lavery, L.A., et al. Risk Factors for Developing Osteomyelitis in Patients with Diabetic Foot Wounds. Diabetes Research and Clinical Practice 83(3):347-52, 2009.
55. Holstein, P., et al. Decreasing Incidence of Major Amputations in People with Diabetes. Diabetologia 43(7):844-7, 2000.
56. Patout, C.A., Jr., et al. Effectiveness of a Comprehensive Diabetes Lower-Extremity Amputation Prevention Program in a Predominantly Low-Income African-American Population. Diabetes Care 23(9):1339-42, 2000.
57. Pinzur, M.S., Slovenkai, M.P., Trepman, E. Guidelines for Diabetic Foot Care. The Diabetes Committee of the American Orthopaedic Foot and Ankle Society. Foot & Ankle International 20(11):695-702, 1999.
58. https://www.acfas.org/footankleinfo/diabetic-guidelines.htm. Last accessed June 17, 2019.
59. Kim, P.J., et al. Critical Elements to Building an Effective Wound Care Center. Journal of Vascular Surgery 57(6):1703-9, 2013.
60. https://www.medicare.gov/coverage/therapeutic-shoes-inserts#. Last accessed June 17, 2019.
61. Lavery, L.A., et al. Shear-Reducing Insoles to Prevent Foot Ulceration in High-Risk Diabetic Patients. Advances in Skin & Wound Care 25(11):519-24; quiz 525-6, 2012.
62. Viswanathan, V., et al. Effectiveness of Different Types of Footwear Insoles for the Diabetic Neuropathic Foot: A Follow-Up Study. Diabetes Care 27(2):474-7, 2004.
63. Busch, K., Chantelau, E. Effectiveness of a New Brand of Stock 'Diabetic' Shoes to Protect against Diabetic Foot Ulcer Relapse. A Prospective Cohort

Study. Diabetic Medicine 20(8):665-9, 2003.
64. Bus, S.A., et al. Effect of Custom-Made Footwear on Foot Ulcer Recurrence in Diabetes: A Multicenter Randomized Controlled Trial. Diabetes Care 36(12):4109-16, 2013.
65. Lavery, L.A., et al. Diabetic Foot Prevention: A Neglected Opportunity in High-Risk Patients. Diabetes Care 33(7):1460-2, 2010.
66. Reiber, G.E., et al. Effect of Therapeutic Footwear on Foot Reulceration in Patients with Diabetes: A Randomized Controlled Trail. JAMA 287(19):2552-8, 2002.
67. Uccioli, L., et al. Manufactured Shoes in the Prevention of Diabetic Foot Ulcers. Diabetes Care 18(10):1376-8, 1995.
68. Ulbrecht, J.S., et al. Prevention of Recurrent Foot Ulcers with Plantar Pressure-Based In-Shoe Orthoses: The CareFUL Prevention Multicenter Randomized Controlled Trial. Diabetes Care 37(7):1982-9, 2014.
69. Rizzo, L., et al. Custom-Made Orthesis and Shoes in a Structured Follow-Up Program Reduces the Incidence of Neuropathic Ulcers in High-Risk Diabetic Foot Patients. The International Journal of Lower Extremity Wounds 11(1):59-64, 2012.
70. Dorresteijn, J.A., et al. Patient Education for Preventing Diabetic Foot Ulceration. Cochrane Database of Systematic Reviews (5):CD001488, 2010.
71. Lavery, L.A., et al. Preventing Diabetic Foot Ulcer Recurrence in High-Risk Patients: Use of Temperature Monitoring as a Self-Assessment Tool. Diabetes Care 30(1):14-20, 2007.
72. Armstrong, D.G., et al. Skin Temperature Monitoring Reduces the Risk for Diabetic Foot Ulceration in High-Risk Patients. American Journal of Medicine 120(12):1042-6, 2007.
73. American Diabetes Association. 2. Classification and Diagnosis of Diabetes. Diabetes Care 40(Suppl 1): S11-S24, 2017.
74. Steed, D.L., et al. Effect of Extensive Debridement and Treatment on the Healing of Diabetic Foot Ulcers. Diabetic Ulcer Study Group. Journal of the American College of Surgeons 183(1):61-4, 1996.
75. Steed, D.L., The Wound Healing Society (WHS) Evaluation of the Science to Arrive at Guidelines. Wounds 13(5 Suppl E):15E-16E, 2001.
76. Cornell, R.S., et al. Debridement of the Noninfected Wound. Journal of Vascular Surgery 52(3 Suppl):31S-36S, 2010.
77. Armstrong, D.G., et al. Maggot Therapy in “Lower-Extremity Hospice” Wound Care: Fewer Amputations and More Antibiotic-Free Days. Journal of the American Podiatric Medical Association 95(3):254-7, 2005.
78. Sherman, R.A. Maggot Therapy for Treating Diabetic Foot Ulcers Unresponsive to Conventional Therapy. Diabetes Care 26(2):446-51, 2003.
79. Lavery, L.A., et al. Total Contact Casts: Pressure Reduction at Ulcer Sites and the Effect on the Contralateral Foot. Archives of Physical Medicine and Rehabilitation 78(11):1268-71, 1997.

80. Frykberg, R.G. A Summary of Guidelines for Managing the Diabetic Foot. Advances in Skin & Wound Care 18(4):209-14, 2005.
81. Lavery, L.A., et al. Reducing Dynamic Foot Pressures in High-Risk Diabetic Subjects with Foot Ulcerations. A Comparison of Treatments. Diabetes Care 19(8):818-21, 1996.
82. Lavery, L.A., Armstrong, D.G., Walker, S.C. Healing Rates of Diabetic Foot Ulcers Associated with Midfoot Fracture due to Charcot's Arthropathy. Diabetic Medicine 14(1):46-9, 1997.
83. Armstrong, D.G., et al. Off-Loading the Diabetic Foot Wound: A Randomized Clinical Trial. Diabetes Care 24(6):1019-22, 2001.
84. Sinacore, D.R. Total Contact Casting for Diabetic Neuropathic Ulcers. Physical Therapy 76(3):296-301, 1996.
85. Caputo, G.M., Ulbrecht, J.S., Cavanagh, P.R. The Total Contact Cast: A Method for Treating Neuropathic Diabetic Ulcers. American Family Physician 55(2):605-11, 615-6, 1997.
86. Veves, A., et al. Graftskin, a Human Skin Equivalent. Is Effective in the Management of Noninfected Neuropathic Diabetic Foot Ulcers: A Prospective Randomized Multicenter Clinical Trial. Diabetes Care 24(2):290-5, 2001.
87. Veves, A., Sheehan, P., Pham, H.T. A Randomized, Controlled Trial of Promogran (a Collagen/Oxidized Regenerated Cellulose Dressing) vs Standard Treatment in the Management of Diabetic Foot Ulcers. Archives of Surgery 137(7):822-7, 2002.
88. Knowles, E.A., et al. Offloading Diabetic Foot Wounds using the Scotchcast Boot: A Retrospective Study. Ostomy/Wound Management 48(9):50-3, 2002.
89. Armstrong, D.G., et al. Evaluation of Removable and Irremovable Cast Walkers in the Healing of Diabetic Foot Wounds: A Randomized Controlled Trial. Diabetes Care 28(3):551-4, 2005.
90. Sibbald, R.G., Woo, K., Ayello, E.A. Increased Bacterial Burden and Infection: The Story of NERDS and STONES. Advances in Skin & Wound Care 19(8):447-61; quiz 461-3, 2006.
91. McGuire, J.B. Pressure Redistribution Strategies for the Diabetic or At-Risk Foot: Part II. Advances in Skin & Wound Care 19(5):270-7; quiz 277-9, 2006.
92. McGuire, J. Transitional Off-Loading: An Evidence-Based Approach to Pressure Redistribution in the Diabetic Foot. Advances in Skin & Wound Care 23(4): 175-88; quiz 189-90, 2010.
93. Armstrong, D.G., et al. Validation of a Diabetic Foot Surgery Classification. International Wound Journal 3(3):240-6, 2006.
94. Katz, I.A., et al. A Randomized Trial of Two Irremovable Off-Loading Devices in the Management of Plantar Neuropathic Diabetic Foot Ulcers. Diabetes Care 28(3):555-9, 2005.
95. Services, D.o.H.a.H. Sandals. London, UK: Health Resources and Services Administration, Revised April 2017.
96. Lair, G. Use of the Ipos Shoe in the Management of Patients with Diabetes Mellitus. Cleveland, OH: Cleveland Clinic Foundation, 1992.

97. Myerson, M., et al. The Total-Contact Cast for Management of Neuropathic Plantar Ulceration of the Foot. The Journal of Bone and Joint Surgery. American Volume 74(2):261-9, 1992.
98. Armstrong, D.G., Lavery, L.A. Negative Pressure Wound Therapy after Partial Diabetic Foot Amputation: A Multicentre. Randomised Controlled Trial. Lancet 366(9498):1704-10, 2005.
99. Saxena, V., et al. Vacuum-Assisted Closure: Microdeformations of Wounds and Cell Proliferation. Plastic and Reconstructive Surgery 114(5):1086-96; discussion 1097-8, 2004.
100. Lehner, B., et al. First Experiences with Negative Pressure Wound Therapy and Instillation in the Treatment of Infected Orthopaedic Implants: A Clinical Observational Study. International Orthopaedics 35(9): 1415-20, 2011.
101. Kim, P.J., et al. The Impact of Negative-Pressure Wound Therapy with Instillation Compared with Standard Negative-Pressure Wound Therapy: A Retrospective, Historical, Cohort, Controlled Study. Plastic and Reconstructive Surgery 133(3):709-16, 2014.
102. Kim, P.J., et al. Negative-Pressure Wound Therapy with Instillation: International Consensus Guidelines. Plastic and Reconstructive Surgery 132(6):1569-79, 2013.
103. Szuminsky, N.J., et al. Effect of Narrow, Pulsed High Voltages on Bacterial Viability. Physical Therapy 74(7):660-7, 1994.
104. Sakmann, B., Neher, E. Patch Clamp Techniques for Studying Ionic Channels in Excitable Membranes. Annual Review of Physiology 46:455-72, 1984.
105. Soong, H.K., et al. Movements of Cultured Corneal Epithelial Cells and Stromal Fibroblasts in Electric Fields. Investigative Ophthalmology & Visual Science 31(11):2278-82, 1990.
106. Zhao, M. Electrical Fields in Wound Healing—An Overriding Signal that Directs Cell Migration. Seminars in Cell and Developmental Biology 20(6):674-82, 2009.
107. Peters, E.J., et al. The Benefit of Electrical Stimulation to Enhance Perfusion in Persons with Diabetes Mellitus. Journal of Foot and Ankle Surgery 37(5):396-400; discussion 447-8, 1998.
108. Xu, L., et al. Bacterial Load Predicts Healing Rate in Neuropathic Diabetic Foot Ulcers. Diabetes Care 30(2):378-80, 2007.
109. Kincaid, C.B., Lavoie, K.H. Inhibition of Bacterial Growth In Vitro Following Stimulation with High Voltage, Monophasic, Pulsed Current. Physical Therapy 69(8):651-5, 1989.
110. Wunderlich, R.P., Peters, E.J., Lavery, L.A. Systemic Hyperbaric Oxygen Therapy: Lower-Extremity Wound Healing and the Diabetic Foot. Diabetes Care 23(10):1551-5, 2000.
111. Londahl, M., et al. Hyperbaric Oxygen Therapy Facilitates Healing of Chronic Foot Ulcers in Patients with Diabetes. Diabetes Care 33(5):998-1003, 2010.
112. Fernandez-Montequin, J.I., et al. Intra-Lesional Injections of Recombinant Human Epidermal Growth Factor Promote Granulation and Healing in

Advanced Diabetic Foot Ulcers: Multicenter, Randomised, Placebo-Controlled, Double-Blind Study. International Wound Journal 6(6):432-43, 2009.
113. Game, F.L., et al. A Systematic Review of Interventions to Enhance the Healing of Chronic Ulcers of the Foot in Diabetes. Diabetes/Metabolism Research and Reviews 28(Suppl 1):119-41, 2012.
114. Lipsky, B.A. Evidence-Based Antibiotic Therapy of Diabetic Foot Infections. FEMS Immunology and Medical Microbiology 26(3-4):267-76, 1999.
115. Ryssel, H., et al. The Antimicrobial Effect of Acetic Acid—An Alternative to Common Local Antiseptics? Burns 35(5):695-700, 2009.
116. Koburger, T., et al. Standardized Comparison of Antiseptic Efficacy of Triclosan, Pvp-Iodine, Octenidine Dihydrochloride, Polyhexanide and Chlorhexidine Digluconate. Journal of Antimicrobial Chemotherapy 65(8):1712-9, 2010.
117. Huyette, D.R., et al. Eradication by Surfactant Irrigation of *Staphylococcus aureus* from Infected Complex Wounds. Clinical Orthopaedics and Related Research (427):28-36, 2004.
118. Conroy, B.P., et al. Comparison of Castile Soap, Benzalkonium Chloride, and Bacitracin as Irrigation Solutions for Complex Contaminated Orthopaedic Wounds. Journal of Orthopaedic Trauma 13(5):332-7, 1999.
119. Anglen, J.O. Comparison of Soap and Antibiotic Solutions for Irrigation of Lower-Limb Open Fracture Wounds. A Prospective, Randomized Study. Journal of Bone and Joint Surgery 87(7):1415-22, 2005.
120. Litzelman, D.K., et al. Reduction of Lower Extremity Clinical Abnormalities in Patients with Non-Insulin-Dependent Diabetes Mellitus. A Randomized, Controlled Trial. Annals of Internal Medicine 119(1):36-41, 1993.
121. Donohoe, M.E., et al. Improving Foot Care for People with Diabetes Mellitus—A Randomized Controlled Trial of an Integrated Care Approach. Diabetic Medicine 17(8):581-7, 2000.
122. Peters, E.J., Lavery, L.A.; International Working Group on the Diabetic Foot. Effectiveness of the Diabetic Foot Risk Classification System of the International Working Group on the Diabetic Foot. Diabetes Care 24(8):1442-7, 2001.
123. Rosenblum, B.I., et al. Preventing Loss of the Great Toe with the Hallux Interphalangeal Joint Arthroplasty. Journal of Foot and Ankle Surgery 33(6): 557-60, 1994.
124. Armstrong, D.G., et al. Is Prophylactic Diabetic Foot Surgery Dangerous? Journal of Foot and Ankle Surgery 35(6):585-9, 1996.
125. Armstrong, D.G., et al. Clinical Efficacy of the First Metatarsophalangeal Joint Arthroplasty as a Curative Procedure for Hallux Interphalangeal Joint Wounds in Patients with Diabetes. Diabetes Care 26(12):3284-7, 2003.
126. Armstrong, D.G., et al. Lengthening of the Achilles Tendon in Diabetic Patients Who Are at High Risk for Ulceration of the Foot. The Journal of Bone and Joint Surgery. American Volume 81(4):535-8, 1999.

127. Mueller, M.J., et al. Effect of Achilles Tendon Lengthening on Neuropathic Plantar Ulcers. A Randomized Clinical Trial. The Journal of Bone and Joint Surgery 85-A(8):1436-45, 2003.
128. Lin, S.S., Lee, T.H., Wapner, K.L. Plantar Forefoot Ulceration with Equinus Deformity of the Ankle in Diabetic Patients: The Effect of Tendo-Achilles Lengthening and Total Contact Casting. Orthopedics 19(5):465-75, 1996.
129. Laborde, J.M. Neuropathic Toe Ulcers Treated with Toe Flexor Tenotomies. Foot & Ankle International 28(11):1160-4, 2007.
130. Lin, S.S., Bono, C.M., Lee, T.H. Total Contact Casting and Keller Arthoplasty for Diabetic Great Toe Ulceration under the Interphalangeal Joint. Foot & Ankle International 21(7):588-93, 2000.
131. American Association of Diabetes Educators. What You Say Matters. 2019. Available at: www.diabeteseducator.org/practice/educator-tools/diabetes-language-paper
132. American Association of Diabetes Educators. Speaking the Language of Diabetes: Language Guidance for Diabetes-Related Research, Education, and Publications. 2017. Available at: www.diabeteseducator.org/docs/default-source/practice/educator-tools/HCP-diabetes-language-guidance.pdf?sfvrsn=8
133. Dickinson, J.K., Guzman, S.J., Maryniuk, M.D., et al. The Use of Language in Diabetes Care and Education. The Diabetes Educator 40(12):1790-1799, 2017.
134. Dickinson, J.K., Guzman, S.J., Maryniuk, M.D., et al. The Use of Language in Diabetes Care and Education. The Diabetes Educator 43(6):551-564, 2017.
135. Dickinson, J.K., Funnell, M.M. Diabetes: Changing the Conversation. Nursing 49(6):57-60, 2019.
136. Funnell, M.M. Patient Empowerment: What Does It Really Mean? Patient Education and Counseling 99(12):1921-2, 2016.

19

Sickle Cell Ulcers

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Objectives

After completing this chapter, you'll be able to:

- understand the pathogenesis of sickle cell anemia (or sickle cell disease)
- discuss the pathogenesis of sickle cell ulcers
- differentiate sickle cell ulcers from arterial and venous ulcers
- implement protocols for prevention and treatment of complications of sickle cell ulcers.

Sickle Cell Disease

Sickle cell ulcers are a complication of sickle cell disease, an inherited genetic disorder of the oxygen-carrying hemoglobin in red blood cells. Sickle cell anemia (or sickle cell disease) was first reported in 1910 by Dr. J.B. Herrick.[1] It is the result of a mutation on the β-globin gene of the cellular DNA. This causes an abnormal amino acid, valine, instead of glutamic acid to be inserted into the β-globin chain of the normal hemoglobin unit resulting in hemoglobin S or sickle hemoglobin.[2–6] This abnormality results in the polymerization of the hemoglobin when it is deoxygenated and a subsequent loss of water from the cell. This polymerization and dehydration of the cells lead to the distortion and sickle shape of the red blood cells and damage to the cell membranes.[3,6] These abnormalities result in occlusion of small blood vessels and capillaries, causing ischemia of the tissues served by these vessels.

Sickle cell disease is primarily seen in black individuals and is more prevalent in the United States, Africa, and the Caribbean countries. The disease is seen in two main forms: (a) homozygous sickle cell disease, the most severe form, occurring when the individual receives a gene for the abnormal hemoglobin (hemoglobin S) from both the mother and the father, and (b) heterozygous sickle cell disease, the less severe form, occurring when the individual receives only one gene for the abnormal hemoglobin from either the mother or father and the other gene is for normal hemoglobin.

Prevalence and Incidence

The patient with homozygous form of sickle cell disease is most likely to develop a sickle cell ulcer. Studies have shown that males are more likely to develop leg ulcers due to sickle cell disease than females.[7] The same study found that 5% of males with sickle cell disease who were over age 10 had sickle cell ulcers[7] and 75% of patients over age 30 had a sickle cell ulcer at some time during the course of their disease.[8] With over 100,000 patients in the United States with sickle cell disease,[9] this makes the number of patients with sickle cell ulcers significant. It is estimated that[9]:

- SCD occurs among about 1 out of every 365 Black or African American births.
- SCD occurs among about 1 out of 16,300 Hispanic American birth.
- About 1 in 13 Black or African American babies is born with sickle cell trait (SCT).

Practice Point

Lower extremity ulcers in young black patients, especially males, could be due to undiagnosed sickle cell disease.

Ulcer Pathogenesis

The abnormal hemoglobin molecule in the red blood cell in the patient with sickle cell disease does not affect the amount of oxygen the red blood cell can carry. After the red blood cell and its hemoglobin give up the oxygen to the tissues, the abnormal hemoglobin causes the red blood cell to lose its water content and polymerize resulting in the red blood cell distorting, becoming rigid, and deforming into the sickle shape (Fig. 19-1). Unfortunately, while the cells are in the sickled shape, they tend to increase blood viscosity and become “sticky.” This results in slowing of the blood flow in small vessels and subsequent clotting of the vessels especially in the postcapillary venules,[6] causing ischemia of tissues and organs. Over time, the patient suffers repeated episodes of pain, episodes of ischemia and reperfusion, and chronic inflammation with subsequent tissue damage and, eventually, organ failure.[5] Because the cells become damaged while they are in the sickled shape, they are removed from the circulation faster than normal, resulting in anemia (Table 19-1). When the red blood cell is reoxygenated and regains its water content, it can regain its normal shape, but a significant percentage of sickle cells do not regain their normal shape and remain irreversibly sickled despite being fully oxygenated.[6] This leads to an even more rapid removal of these irreversibly damaged cells from the circulation.

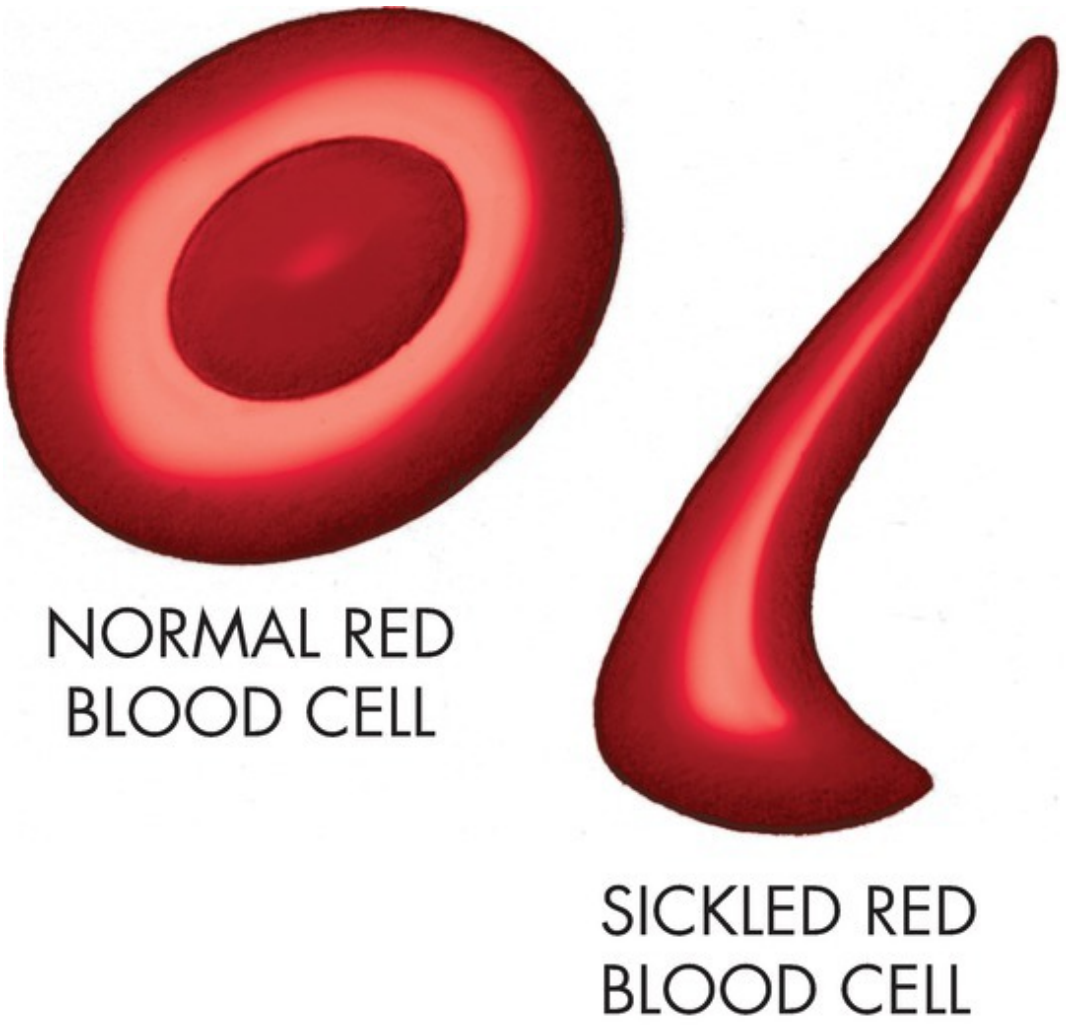

Figure 19-1 Sickled cell. The diagrams show a normal red blood cell and a sickled cell.

Table 19-1 Conditions Associated with Sickle Cell Anemia

Complication	Cause
"Crisis" with fever and pain	Sickling of cells due to abnormal hemoglobin
Pain in bones, joints, and back	Sickling of cells and ischemia of tissues
Severe abdominal pain	Sickling of cells and ischemia of tissues
Pregnancy problems Fertility problems	Uncontrolled sickling of cells
Increased infections Pulmonary Urinary tract	Deficient immune response
Salmonella osteomyelitis	Ischemia of bones, bone infarcts, sepsis
Chronic leg ulcers	Sickling of cells and ischemia of tissues
"Hand–foot" syndrome	Sickling of cells and ischemia of bones
Avascular necrosis of femoral or humeral head	Ischemic necrosis of bones due to sickling
Visual problems	Ischemia of retina due to sickling
Pulmonary infarction	Ischemia of lung due to sickle cell emboli
Congestive heart failure Cardiac murmurs EKG abnormalities	Myocardial ischemia
Jaundice	Hemolytic anemia Gallstone production and obstructive jaundice
Cirrhosis of liver	Ischemia of liver and cell necrosis
Hepatitis	Multiple blood transfusions
Enlarged spleen (infancy only)	Increased blood production
Splenic infarction (late teens, adult)	Ischemia of tissue due to sickling of cells
Renal dysfunction Hematuria	Ischemia of kidney with infarction of tissue
Renal vein thrombosis	Sickling of cells
Priapism (especially in children)	Sickling of cells
Impotence	Damage of penis by priapism and ischemia
Anemia	Hemolysis of abnormal cells
"Aplastic crisis"	Failure of bone marrow to produce cells due to infarction of marrow
Folate deficiency	High folate requirement of hemolytic anemia

Adapted from Conley, C.L. "The Hemoglobinopathies and Thalassemias." In P.B. Beeson, W. McDermott, eds. *Textbook of Medicine*, 13th ed. Philadelphia, PA: W.B. Saunders Co., 1971:1501-3, with permission.

Although the underlying pathophysiology remains ischemia due to occlusion of the microcirculation, sickle cell ulcers have been associated with homozygous hemoglobin SS disease, trauma, infection, severe anemia, warm temperatures,[10,11] venous

insufficiency,[12,13] and indications of severe sickle cell disease as reflected by increased cardiovascular risk, pulmonary hypertension, and lower body mass index.[14]

These ulcers are most likely to occur in the malleolar area of the lower extremities with the medial malleolar area being the most commonly involved.[15] Occlusion of the small vessels in the microcirculation results in chronic damage to the microcirculation of the skin around the ankle including injury to the capillary walls, thickening of the intimal lining of the capillary, and an increase in permeability of the vessel wall, allowing macromolecules to escape into the tissues.[16–18] These changes can also result in a chronic inflammatory condition and ischemia of the tissues making them more susceptible to minor trauma and less able to heal.[16,19] This makes these areas more likely to be the sites of skin breakdown and ulceration. The repeated cycle of ischemia then reperfusion results in a marked inflammatory reaction in the tissues furthering the tissue damage caused by the ischemia alone. It has been found that the endothelial dysfunction in sickle cell disease may be worsened due to impaired bioavailability of endogenous nitric oxide. The red blood cell hemolysis that results from the damage to the cells releases hemoglobin into the circulation binding the nitric oxide preventing it from dilating the blood vessels.[20] This results in unrestrained vasoconstriction of the small vessels, ischemia of the skin, pain, and skin necrosis.[19,21]

Diagnosis

Medical History

Evaluation of the patient with a suspected sickle cell ulcer is of utmost importance so that the correct diagnosis can be made and appropriate treatment planned. The patient's medical history should be recorded, and the events surrounding the ulcer development should be investigated. Is this the first ulcer the patient has had? How long has the ulcer been present? How did the ulcer first develop? Was there trauma to the area? How did the area first look? Any history of lower extremity edema; unexplained swelling of the hands, feet, or knees; osteomyelitis; episodes of abdominal or joint pain; episodes of severe unexplained pain; recurrent urinary tract infections or pneumonia; or anemia should be noted. Sickle cell patients are prone to develop unexplained episodes of fever, which tend to resolve without therapy. These patients may carry a diagnosis of fever of unknown origin (FUO).

Physical Examination

A complete physical examination should be part of the evaluation of the patient. Vital signs, especially temperature, should be taken because, as mentioned above, unexplained fever may be a sign of sickle cell disease. Cardiac and pulmonary exam may reveal signs of cardiovascular disease and pulmonary hypertension. These conditions are more frequent and more severe in sickle cell patients with ulcers than in those without ulcers.[14] Abdominal examination can detect enlargement of the liver. The spleen may be enlarged in early childhood as it is a primary blood-forming organ but is usually small in later life due to ischemic infarction.

The presence of scars on the extremities indicates previous ulcer incidence. The location of the ulcer or ulcers is important as most sickle cell ulcers are found on the lower one-third of the leg and usually over the malleoli of the ankle but are more frequent over the medial malleoli[11,15]; however, less common sites include any area with little subcutaneous tissue such as the anterior tibial area, dorsum of the foot, and the Achilles tendon area.[22] The size of each ulcer should be measured by determining the length and width or by using one of the more advanced measuring modalities described elsewhere in the book. (See Chapter 6, Wound Assessment.)

The presence of an ulcer in a patient with varicose veins, venous insufficiency, and sickle cell disease can be especially troublesome as it may lead to misdiagnosis (Fig. 19-2A and B). In addition, venous incompetence in the patient with sickle cell disease may predispose him to develop an ulcer and can be correlated with the development of a recurrent sickle cell ulcer.[13]

A

B

Figure 19-2 Undiagnosed sickle cell ulcer treated initially as a "venous ulcer." **(A)** Predebridement. **(B)** Postdebridement. (Used with permission from T. Treadwell, MD.)

Noninvasive venous studies have shown that venous insufficiency and venous hypertension may be associated with the development and recurrence of sickle cell ulcers.[22,23] As a result, treatment of venous disease should play a part in the correct approach to therapy of patients with sickle cell ulcers.

Practice Point

Misdiagnosis—and thus, mistreatment— of sickle cell ulcer as a venous "stasis" ulcer makes it imperative to get the diagnosis correct.

Ulcer Assessment

Examination of the wound bed is essential to determine the presence of granulation tissue or fibrinous material (“slough”) (Figs. 19-2A and B and 19-3A and B). In addition, the presence of periulcer erythema or cellulitis should also be documented. The presence and character of any drainage should be noted. Tenderness of the lower extremity to palpation or the presence of pain in the ulcer or surrounding area should be also recorded. A vascular examination should always be performed by checking for the dorsalis pedis and posterior tibial pulses to be sure the patient has adequate blood flow. Documentation of their presence or absence is important. If there is a question about the adequacy of the circulation, the patient will need to be referred for noninvasive vascular studies or arteriography. The microcirculation in the periwound area can be evaluated with pulse oximetry, laser Doppler, and $TcPO_2$ measurements. Measurements of tissue oxygen levels by whatever method showed that readings are lower in patients with ulcers than sickle cell patients without ulcers.[14]

A

B

Figure 19-3 Recurrent sickle cell ulcer treated with tissue-engineered skin. **(A)** Healed 8 weeks postapplication. **(B)** Ulcer remains healed 8 months later—note improved scar. (Used with permission from T. Treadwell, MD.)

Practice Point

A complete history and physical examination are vital when evaluating a patient with a sickle cell ulcer.

Laboratory Assessment

Laboratory evaluation can help make the diagnosis if a patient is seen with a nonhealing ulceration of the ankle area. It, also, will help identify the severity of the patient's sickle cell disease. If the patient does not have a diagnosis of sickle cell disease, but it is suspected, blood tests that check for anemia, sickle cells, abnormal hemoglobin, and other hemolytic anemias should be ordered. This usually involves a complete blood count (CBC) with platelet count, sickle "prep," and a hemoglobin electrophoresis. Although hemoglobin electrophoresis is considered the diagnostic tool of choice, it has its limitations especially if the patient has had recent blood transfusions.[10] In such a case, referral to a hematologist may be necessary. If the patient with known sickle cell disease has an ulcer, the laboratory workup should consist of a CBC with differential white blood cell count, platelet count, and reticulocyte count.

Laboratory evaluation has shown that sickle cell ulcer patients, in addition to lower hemoglobin levels, have higher markers of hemolysis such as elevated levels of lactate dehydrogenase (LDH), bilirubin, aspartate transaminase (AST), alkaline phosphatase, and uric acid, serum amino-terminal probrain-type natriuretic peptide, and higher reticulocyte counts than do patients with sickle cell disease with no ulcers.[14,16] High ferritin levels are noted and are felt to be due to the chronic inflammatory state and chronic hemolysis in these patients with ulcers.[14] High levels of uric acid are seen in patients with sickle cell ulcers. It has been determined that high uric acid levels may be an indicator of microvascular problems and hemolysis even if there is no current sickle cell ulcer.[14]

Patients with sickle cell disease are especially prone to develop infections; therefore, it is important to obtain a wound culture using appropriate technique if the wound bioburden appears elevated. We have found that a significant number of patients with sickle cell ulcers have wounds covered by biofilm, which must be removed before the ulcer can heal (see Fig. 19-2A and B). The most frequent way to remove a biofilm is with sharp debridement, but recently, we have turned to ultrasonic debridement techniques, enzymatic debridement,

and topical medications designed specifically for the treatment of biofilms, which are less painful for the patient. Dressings that enhance autolytic debridement can, also, be helpful. Following debridement, the use of the enzymatic agent, collagenase, will help maintain the debrided state.[24] Biofilm is covered in another section of this book. (See Chapter 7, Wound Bioburden and Infection.)

Infection and Osteomyelitis

Patients with sickle cell disease and a deep, painful ulcer should be evaluated radiologically for the presence of osteomyelitis especially if the patient has fever or leukocytosis. Patients with sickle cell disease are prone to developing Salmonella osteomyelitis.[10] Radiologic evaluation can be done by several methods. Plain film x-rays are the least sensitive method and usually don't show any evidence of osteomyelitis until late in the course of the infection. In addition, they can be confusing in the patient with sickle cell disease as the disease can cause periosteal elevation and other bone changes mimicking osteomyelitis.[10] Nuclear medicine bone scans are slightly more helpful, but it must be remembered that routine bone scans only detect areas of inflammation. For this reason, if the patient has an ulcer overlying the bone in question, the bone scan is not helpful without special imaging techniques. Magnetic resonance imaging appears to be the imaging modality of choice in terms of sensitivity and specificity.

It has been suggested that bone biopsy and culture may be the only definitive way to determine if osteomyelitis is present,[11] but it should be done with great care so as not to cause an infection in the bone. Biopsy of the wound bed and wound margin may be advisable if the ulcer has been present for over 3 months, does not respond to therapy, or just doesn't "look right." This should be done to rule out the possibility of malignancy and can help with the diagnosis.[25]

Pain

It must be remembered that sickle cell ulcers are extremely painful. Pain assessment is an area that must not be overlooked as, often times, the patient is fearful of experiencing even more pain with your evaluation and treatment and may not want you to look at the ulcer much less touch it. This makes treatment of these ulcers very difficult. (A useful pain assessment tool for evaluating a patient's pain is outlined elsewhere in this book [see Chapter 12, Pain Management and Wounds].) It has been the author's experience that if the provider

does not address the pain problem, many of the patients will not return for follow-up care. Indeed, most patients would rather keep their ulcer than deal with potentially being in more pain than they already are. For this reason, any procedure to the wound must be preceded by some type of anesthesia: topical anesthesia (Xylocaine ointment or EMLA cream), injectable local anesthesia, regional anesthesia, or general anesthesia. Some of these techniques will require hospitalization of the patient.

Pain control can be managed with topical anesthetic agents. Topical Xylocaine ointment or EMLA cream can be used on a regular basis for pain control. Applied every 4 to 6 hours, these agents can make the patient's daily activities much more manageable. It also makes dressing changes more comfortable. The use of topical opioid medications has recently been useful in treating painful sickle cell leg ulcers without some of the complications seen with systemic opioids.[26,27] Other therapies, including oral pain medications and regional medications such as Xylocaine patches, are useful but many times should be managed by a pain specialist. (See Chapter 12, Pain Management and Wounds.)

Because of the painful nature of sickle cell ulcers, debridement, biopsy cultures, or even dressing changes can be a challenge. If biopsy cultures are indicated, pain management will be required, but the practitioner should be aware of studies about the effects of anesthetics on bacteria. Berg et al.[28] have shown that EMLA cream, a topical anesthetic agent commonly used before doing wound biopsies, is highly antibacterial. Within 1 hour of exposure to EMLA cream, most common bacteria are killed, including strains of *Staphylococcus aureus* (both methicillin-resistant and methicillin-sensitive strains), *Streptococcus pyogenes*, *Escherichia coli*, and *Pseudomonas aeruginosa.* Injected solutions of 1% lidocaine are, also, antibacterial for the same organisms but at greater than 2 hours after local injection. It is the recommendation of the author that EMLA cream not be used for anesthesia when biopsy cultures are being done. Local injection of preservative-free 1% lidocaine would be satisfactory to use if the biopsy culture is done within 2 hours of the injection and the specimens are promptly plated.[28]

Practice Point

Sickle cell ulcers are extremely painful. Evaluation and treatment of the patient's pain should be a top priority and should be the first steps in instituting therapy for the ulcer.

Treatment

Treatment of sickle cell ulcers can be challenging and frustrating. Even in this day of evidence-based therapies, it is noteworthy that there are no published trials of treatments of sickle cell ulcers.[11] One of the more interesting findings about the treatment of sickle cell ulcers is that most ulcers will heal with prolonged bed rest.[11] This is one of the reasons that stirred researchers to consider the association of venous insufficiency and sickle cell ulcers. Prolonged bed rest relieves venous hypertension and the inflammation associated with it providing a better healing environment for the ulcer.[13,29] Obviously, this is not a practical therapy as extended hospitalization is no longer possible, and complete bed rest at home is not realistic. However, any therapy that results in a long period of immobilization, such as from operative intervention, must take into account that the bed rest is healing the ulcer, not necessarily the treatment. With this in mind, one must begin with the basics of what we know constitutes good wound care.

The basics of good wound care include debridement of devitalized tissue, control of infection, assurance of adequate circulation, maintenance of a moist wound environment, and control of the edema in the extremity. The major addition in the treatment of sickle cell ulcers is control of wound pain. Many times, the ulcers are so painful that manipulation of the wound is impossible. Treatment of the wound with topical anesthetics has been previously addressed and must not be overlooked. If the therapy for the wound is painful, most people will forgo your advice and treat the ulcers themselves in ways that do not cause more pain. It is unfortunate that many of these patients are labeled as “noncompliant” when it is a poor choice of therapy by the clinician and not the compliance of the patient that should be in question. Once the pain is controlled, the wound can be debrided and treated, as indicated. The use of dressings that enhance autolytic debridement or enzymatic debridement should be strongly considered. Debridement with sterile maggots can be a consideration and is less painful, but convincing the patient may be difficult.

The evaluation of sickle cell ulcers for infection and the evaluation of the patient for circulation problems have been covered previously. The importance of moist wound care has been known since Winter's publication in 1963.[30] It is now known that wounds treated with wet-to-dry dressings do not heal well and removing a dry dressing that adheres to the wound causes pain and reinjures the wound.[31] Wounds treated by moist wound healing techniques and dressings heal faster, are less painful, and have less scarring than those allowed to dry out. There are numerous wound dressings currently available, which will maintain a moist wound environment. (See Chapter 9, Wound Treatment Options.)

If the wound is felt to be infected, topical antimicrobials should be considered. Oral or IV antibiotics are only indicated if the patient has a leukocytosis, cellulitis, or fever.[32,33] Silver dressings have become popular in the treatment of the wound with a clinically significant bacterial burden (critical colonization) or with frank infection. There are numerous silver dressings available for use on these wounds. It is suggested that once the bacterial burden is under control, the silver dressing should be discontinued and other moisture control dressings used. This is because of the potential for toxicity of the silver to the growing tissues.[34,35] Dressings containing cadexomer iodine are useful in treating wounds critically colonized with bacteria or wounds, which are infected.[36,37]

Since a number of patients with sickle cell ulcers have a low body mass index and are underweight, attention to nutritional factors should be remembered.[14] Restoration of normal protein, vitamin, and electrolyte levels should be considered critical in the treatment of these patients.[38] It has been shown that many patients with sickle cell ulcers are zinc deficient. Some early studies showed that oral zinc sulfate (200 mg three times per day)[39] is helpful in the treatment of patients with sickle cell ulcers.

Practice Point

Always look for edema on the lower extremities of patients with sickle cell disease as it is a critical indicator prior to ulceration.

Another major issue is to manage the edema and the venous insufficiency of the extremity associated with the ulcer. It is known that edema fluid inhibits mitogenic activity and DNA synthesis, the cytokine environment in edema fluid is more proinflammatory, the protease activity is higher in edema fluid, and growth factor levels are decreased in edema fluid. All of these factors contribute to impaired healing of any wound including sickle cell ulcers.[40] Compression bandaging should be considered in the treatment of any patient with a lower extremity ulcer and edema.

The use of the tissue-engineered skin in the treatment of sickle cell ulcers has met with some degree of success.[41] Prior to applying any advanced therapy product, the wound bed must be well prepared, which means assuring that the wound bed has been debrided to remove all necrotic tissue, that infection has been controlled, and that the wound environment has been optimized.[42] To achieve the goal of wound environment optimization, Treadwell et al.[43] recommend pretreating the wound with a protease-modulating agent for 2 to 3 weeks to reduce the abnormal protease levels. These agents include oral or topical doxycycline or an oxidized, regenerated cellulose (ORC) collagen product.[44,45] His early data show that pretreatment of any chronic wound with these protease-modulating agents prior to application of human skin equivalent improves the healing rate.[43] Once treated with the tissue-engineered skin product, the patient can experience significant relief of their pain as well as healing of his/her ulcer (see Fig. 19-3A). It is also of note that sickle cell ulcers treated with tissue-engineered skin seem to have a more “normal” appearing and stable scar (see Fig. 19-3B).

The use of the split-thickness skin graft or pinch grafts to treat patients with sickle cell ulcers may be a reasonable therapeutic alternative. However, the procedures require hospitalization and anesthesia, thus making them less cost-effective. It has also been reported that split-thickness skin grafting has a very low success rate in healing sickle cell ulcers, and in those that do heal, the recurrence rate is very high.[46] Success has been noted with the use of muscle flaps, myocutaneous flaps, and free flaps to cover large lower extremity sickle cell ulcers[47,48] but not uniformly.[49]

A mainstay in the treatment of sickle cell disease is hydroxyurea, which is known to improve symptoms associated with the disease by increasing the levels of fetal hemoglobin (HbF). This results in a significant decrease in the polymerization of hemoglobin S and sickling of the red blood cells.[6] Unfortunately, hydroxyurea is known to cause leg ulcers in patients taking this medication.[50,51] This may be due to the finding that hydroxyl urea exerts an anti-angiogenic effect in areas needing new blood vessels.[52] This can multiply the issues of hypoxia caused by thrombosis of the microvasculature preventing tissue repair and healing. It is considered best to stop the medication to ensure proper healing.[51] Fortunately, it has been found that ulcers related to hydroxyurea therapy respond promptly to treatment with tissue-engineered skin.[53]

Transfusion therapy has been tried in the treatment of patients with sickle cell ulcers who have been resistant to all other therapies. The goal of the therapy is to keep the hematocrit between 30 and 35 volume percent and the percentage of normal hemoglobin (hemoglobin A) greater than 70% of the total.[46] The transfusions are continued until the ulcer heals or for 6 months at which time they are discontinued. Unfortunately, 20% to 30% of patients treated with multiple blood transfusions can develop antibodies to blood products minimizing their use when anemia is profound.[46] The possibility of iron overload with this therapy must be considered.[17]

Another interesting approach to treating sickle cell ulcers has been the use of IV arginine butyrate. The concept is that the arginine butyrate will change the concentration of abnormal hemoglobin in

addition to stimulating immune function and collagen synthesis. It may also reverse the inflammatory microenvironment, which prevents wound healing.[54–57]

Other therapies for sickle cell ulcers include medications, such as pentoxifylline (Trental) and the topical growth factor, molgramostim (GM-CSF)[58]; negative pressure wound therapy; hyperbaric oxygen therapy; and electromagnetic stimulation. Other therapies are still considered experimental, and their utility in treating these patients with difficult wound problems will be determined by future studies.

It has been suggested that strategies for the treatment of sickle cell ulcers should fall into one of the following categories: (a) agents which reduce or prevent sickle cell dehydration, (b) agents which reduce sickle cell–endothelial adhesive events, (c) nitric oxide or nitric oxide–related compounds, or (d) antioxidant agents.[59] These strategies deal with the unhealed ulcer based on the pathophysiology unique to the sickle cell ulcer.

Nitric oxide is a strong vasodilator, which also effects activation of white blood cells, platelets, and endothelial cells. Nitric oxide is produced by endothelial cells in the vasculature from the amino acid, L-arginine. It has been noted that nitric oxide levels are decreased in patients with sickle cell ulcers especially during episodes of crisis.[59–61]

Based on this information, Aslan[19] suggested that sickle cell ulcers may be due to a decrease in microcirculatory smooth muscle nitric oxide with resultant vasoconstriction, tissue ischemia, and necrosis and that nitric oxide supplementation may improve healing of the ulcers or even help prevent them. Because of this information, we have been treating patients with sickle cell ulcers with a combination of L-methylfolate, pyridoxal 5′-phosphate, and methylcobalamin, the combination of which has been shown to reduce vascular homocysteine levels and raise nitric oxide levels in the microcirculation of wound bed and periwound skin compared to the skin away from the ulcer.[61] An increase in nitric oxide in the microcirculation is reflected by vasodilatation and an increase in tissue $TcPO_2$. An example is the patient represented in Figure 19-4.

The “pretreatment” evaluation shows the periwound $TcPO_2$ (No. 1) and the reference point (No. 2) levels of 16.0 and 69.1, respectively. The periwound level is below what would be expected for healing to occur. After 1 week of oral therapy with the medication (“posttreatment”), the periwound $TcPO_2$ has risen to 46.1 (No. 2), while the reference point (No. 1) has stayed essentially the same at 67.2.[62] With this improvement, the wound began to heal. For these reasons, we recommend all of our sickle cell ulcer patients be treated with this therapy. Recently, we have had the opportunity to utilize a device that delivers topical nitric oxide directly to the wound bed to assist in healing. This is the preferable way to deliver the nitric oxide so that systemic effects can be avoided in these patients.[63] It is our impression that the sickle cell ulcers heal faster with these therapies, but the actual healing rates and whether this treatment will reduce the incidence of sickle cells ulcers or their recurrence are yet to be determined.

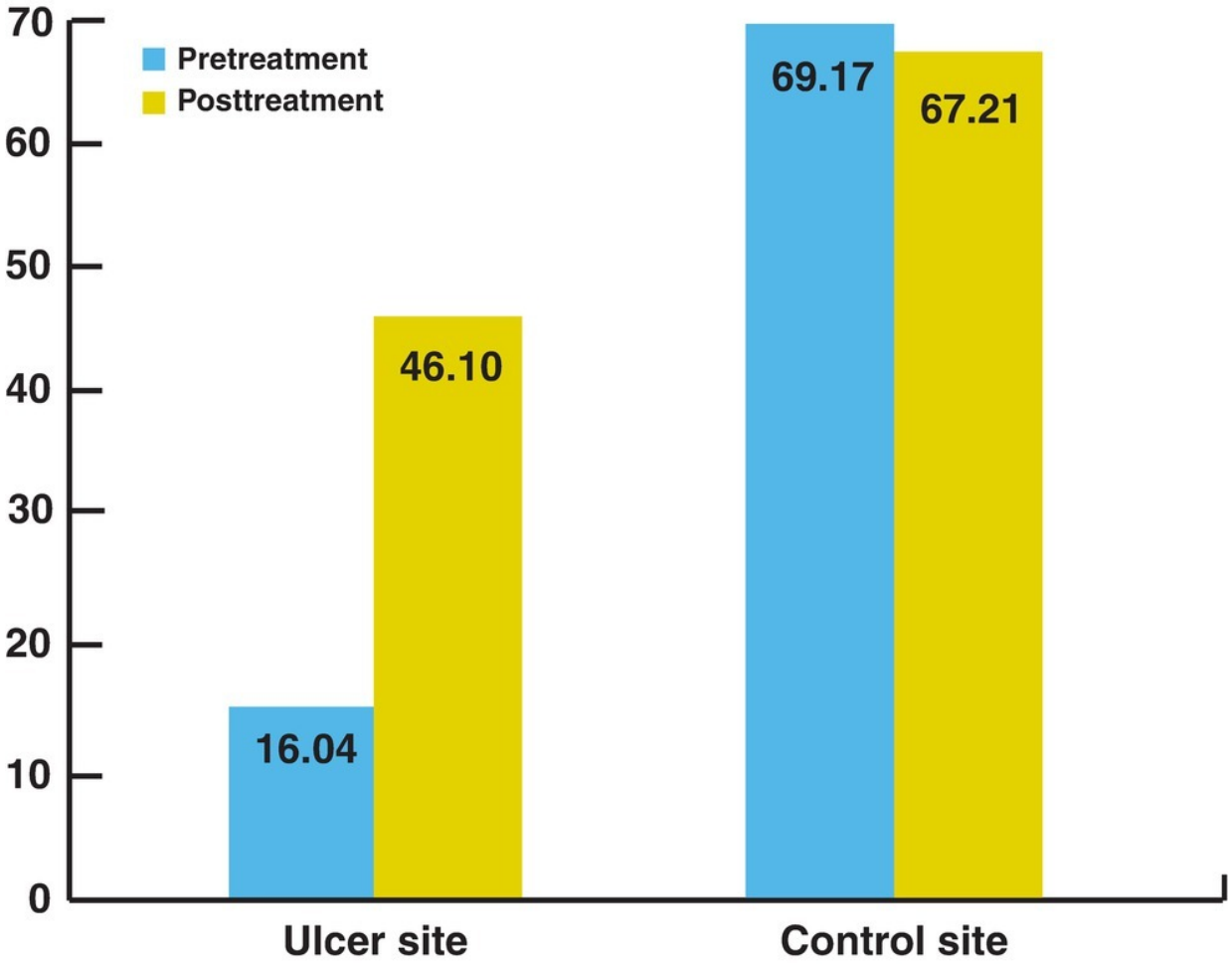

Figure 19-4 Treatment of sickle cell ulcer with nitric oxide–producing medication. Before treatment, $TcPO_2$ levels at the ulcer site and control site were 16.04 and 69.17, respectively. The periwound $TcPO_2$ level is below what would be expected for healing to occur. After 1 week of oral therapy with the nitric oxide–producing medication Metanx, there was marked improvement in the periwound $TcPO_2$, while the control site stayed essentially the same.[32] With this improvement, the wound began to heal. Based on

this, we treat all of our sickle cell ulcer patients with this medication.

The use of immunoglobulins in the treatment of sickle cell ulcers has been recommended. The immunoglobulins can reduce the inflammatory reaction between the cells and improve the blood flow in the microcirculation. It does this by inhibiting the recruitment of neutrophils to the area of the vasoocclusion and by decreasing the adhesiveness of the neutrophils already in the area. This reduces neutrophils adhering to the blood vessel walls and vessel thrombosis. There was experimental evidence that the vasoocclusion in these acute thrombosis episodes may be reversed by the use of immunoglobulins![64] These findings are to be established for clinical effectiveness.

A synthetic heparan sulfate (CACIPLIQ20®) has found to be an effective approach to the treatment of refractory sickle cell ulcers. The resistance of this molecule to protease destruction makes it a longer acting treatment than other similar compounds. It is known to have its effect through its ability to decrease the local inflammatory reaction and stimulate angiogenesis.[65] Another benefit of this product is that the application of the product markedly reduced the patient's wound pain even before healing was noted.[65] Additional investigation of this product is definitely warranted.

Sickle cell ulcers will respond better to any therapy if the sickle cell disease is under control. If the patient's anemia is profound (below 5 g Hg/100 mL) or if the abnormal hemoglobin-containing cells represent greater than 50% of the total volume of red blood cells, the success of any therapy is problematic. This has recently been supported by work reported by Eckman and Platt.[46]

Preventing Ulcers

Prevention of sickle cell ulcers is paramount. Common sense instructions should be given to patients to minimize problems known to be associated with sickle cell ulcer development or recurrence. The patient with sickle cell disease must be instructed in the importance of good skin care and methods to keep the skin moisturized and to avoid trauma to the lower legs and ankles. For example, the patient should use insect repellants to avoid insect bites. It has been noted that edema is the single most common occurrence prior to recurrence of an ulcer.[46] For this reason, the importance of treating lower limb swelling should be emphasized including the use of support hose or elastic wraps. The patient should also be instructed to treat any minor injury to the lower extremities aggressively and seek medical care promptly. The clinician should inspect the lower extremities of sickle cell patients at each visit and review the preventative measures with them routinely.

The key to real prevention of sickle cell ulcers is to remedy the underlying problem—the genetic abnormality that predisposes one to develop an ulcer or any of the other devastating complications associated with this disease. Slowly, with technology, this may be reality. In 2007, Bernaudin and colleagues reported that there had been long-term correction of sickle cell disease with stem cell transplantation.[66] A report in 2017 supported this finding.[67] These studies confirmed the success of ablating the patient's abnormal bone marrow and replacing it with normal blood producing donor bone marrow. This is a great step forward in our managing of this disease. Unfortunately, there has been considerable resistance to using bone marrow transplant for sickle cell patients who currently have a leg ulcer due to the possibility of an overwhelming infection occurring in the immunosuppressed patient. It is with great excitement that reports have been published showing that this is not a problem. Patients with sickle cell disease and ulcers have successfully undergone stem cell transplantation curing their sickle cell disease and their ulcers without catastrophic infection developing from the contaminated wound bed.[68]

This is a great advancement in giving those suffering from this disease hope that a cure may be available for them.

More exciting news has followed with a release from the National Institutes of Health. Six patients treated with a gene therapy product (LentiGlobin®) had reversal of their abnormal hemoglobin production by the bone marrow. This procedure involves treating some of the patient's own bone marrow stem cells with a lentivirus containing normal blood-forming genes. The virus inserts the normal blood-forming genes into the patient's cells. The cells are then given back to the patient to begin producing normal hemoglobin. In the six treated patients, normal hemoglobin in the blood was noted within 3 months of therapy![69] What an exciting development to be able to use the patient's own cells without the risk of a true stem cell transplantation yet with a good outcome!

Summary

Sickle cell ulcers are a potential reality for any person with the inherited disease—sickle cell anemia. Treatment of these ulcers can be frustrating and requires multiple modalities. Unfortunately, recurrence is frequent. With new modalities to treat the underlying disease and the ulcers on the horizon, it is hoped that sickle cell ulcers and the information in this chapter eventually will be of historical interest both here and around the world. In the meantime, understanding of the patient's unique situation and use of evidence-based treatments will result in the best outcomes in the treatment of sickle cells ulcers until a cure is a reality.

Show What You Know

1. **Which of the following is not correct?**
   A. Sickle cell disease is an inherited disease of the blood's hemoglobin molecule.
   B. Sickle cell disease is seen primarily in black individuals.
   C. Higher risk of developing an ulcer is seen in individuals with heterozygous sickle cell disease.
   D. The most severe form of sickle cell disease is seen when an individual inherits an abnormal hemoglobin gene from both parents.

2. **Sickle cell ulcers are more likely to develop in males with sickle cell disease than females.**
   A. True
   B. False

3. **Which one of the following can signal that a patient with sickle cell disease might ulcerate?**
   A. Edema
   B. Hemoglobin A_{1C} of 7
   C. Absence of fever
   D. Resolution of pain in the leg

4. **Which one of the following is not part of the evaluation of the patient with a sickle cell ulcer?**
   A. History including a family history
   B. General physical examination
   C. Assessment of wound bed and pulses
   D. A bone scan

5. **Comprehensive treatment of the patient with sickle cell ulcer could include which of the following?**
   A. Dietary supplements to improve protein, vitamins, and electrolytes
   B. Debridement of the ulcer

C. Wound pain management
D. Compression modalities
E. All of the above

Answers to these questions are found on page 686.

References

1. Herrick, J.B. “Peculiarly Elongated and Sickle-Shaped Red Blood Corpuscles in a Case of Severe Anemia.” Transactions of the Association of American Physicians 25:553, 1910.
2. Eaton, W.A., Hofrichter, J. “Hemoglobin S Gelation and Sickle Cell Disease.” Blood 70:1245-52, 1987.
3. Eaton, W.A., Hofrichter, J. “Sickle Cell Hemoglobin Polymerization.” Advances in Protein Chemistry 40:63-279, 1990.
4. Nagel, R., Platt, O.S. “General Pathophysiology of Sickle Cell Anemia.” In M.H. Steinberg, B.G. Forget, D.R. Higgs, et al., eds. Disorders of Hemoglobin. Cambridge, UK: Cambridge University Press, 2001:494-526.
5. Ladizinski, B., Bazakas, A., Mistry, N., et al. Sickle Cell Disease and Leg Ulcers. Advances in Skin & Wound Care 25:420-8, 2012.
6. DeFranceschi, L., Corrocher, R. “Established and Experimental Treatments for Sickle Cell Disease.” Haematologica 89(3):348-56, 2004.
7. Powars, D.R., Chan, L.S., Hiti, A., et al. “Outcome of Sickle Cell Anemia: A 4-Decade Observational Study of 1056 Patients.” Medicine (Baltimore) 84:363-76, 2005.
8. Charache, S. “One View of the Pathogenesis of Sickle Cell Diseases.” Bulletin Européen de Physiopathologie Respiratoire 19:361-6, 1983.
9. Centers for Disease Control and Prevention. Sickle Cell Disease (SCD) Data and Statistics. Available at: http.www.cdc.gov. Last accessed 10 February 2019.
10. Conley, C.L. “The Hemoglobinopathies and Thalassemias.” In P.B. Beeson, W. McDermott, eds. Textbook of Medicine, 13th ed. Philadelphia, PA: W.B. Saunders Co., 1971:1501-3.
11. Sickle Cell Information Center. “Management of Sickle Cell Disease.” Available at: www.scinfo.org/the-management-of-sickle-cell-disease-4th ed. nih pub.no. 02-2117, Chapter 22. Last accessed May 20, 2015.
12. Cumming, V., King, L., Fraser, R., et al. “Venous Incompetence, Poverty, and Lactic Dehydrogenase in Jamaica Are Important Predictors of Leg Ulceration in Sickle Cell Anemia.” British Journal of Haematology 142:119-25, 2008.
13. Clare, A., FitzHenley, M., Harris, J., et al. “Chronic Leg Ulceration in Homozygous Sickle Cell Disease: The Role of Venous Incompetence.” British Journal of Haematology 119(2):567-71, 2002.
14. Minniti, C.P., Taylor, J.G., Hildesheim, M., et al. “Laboratory and Echocardiography Markers in Sickle Cell Patients with Leg Ulcers.” American Journal of Hematology 86(8):705-8, 2011. doi: 10.1002/ajh.22065.
15. Sawhney, H., Weedon, J., Solomon, G.P., et al. Predilection of Hemolytic Anemia-Associated Leg Ulcers for the Medial Malleolus. VASA 31(3):191-3, 2002.

16. Nolan, V.G., Adewoye, A., Baldwin, C., et al. “Sickle Cell Ulcers: Association with Haemolysis and SNPs in Klotho, TEK and Genes of the TGF-Beta/BMP Pathway.” British Journal of Haematology 133(5):570-8, 2006.
17. Trent, J.T., Kirsner, R.S. “Leg Ulcers in Sickle Cells Disease.” Advances in Skin & Wound Care 17(8):410-6, 2004.
18. Morris, C.R., Kuypers, F.A., Larkin, S., et al. “Arginine Therapy: A Novel Strategy to Induce Nitric Oxide Production in Sickle Cell Disease.” British Journal of Haematology 111:498-500, 2000.
19. Aslan, M., Freeman, B.A. “Oxidant-Mediated Impairment of Nitric Oxide Signaling in Sickle Cell Disease—Mechanisms and Consequences.” Cellular and Molecular Biology 50:95-105, 2004.
20. Kato, G.J., Gladwin, M.T. Evolution of Novel Small-Molecule Therapeutics Targeting Sickle Cell Vasculopathy. JAMA 300:2638-40, 2008.
21. Mack, K.A., Kato, G.J. “Sickle Cell Disease and Nitric Oxide: A Paradigm Shift?” International Journal of Biochemistry & Cell Biology 38(8):1237-43, 2006.
22. Minniti, C.P., Eckman, J., Sebastiani, P., et al. “Leg Ulcers in Sickle Cell Disease.” American Journal of Hematology 85(10):831-3, 2010. Available at: http://wileyonlinelibrary.com/cgi-bin/jhome/35105
23. Mohan, J.S., Vigilance, J.E., Marchall, J.M., et al. Abnormal Venous Function in Patients with Homozygous Sickle Cell (SS) Disease and Chronic Leg Ulcers. Clinical Science 98:667-72, 2000.
24. Falanga, V. “Wound Bed Preparation and the Role of Enzymes: A Case for Multiple Actions of Therapeutic Agents.” Wounds 14(2):47-57, 2002.
25. Ackroyd, J.S., Young, A.E. “Leg Ulcers That Do Not Heal.” British Medical Journal 286:207-8, 1983.
26. Twillman, R.K., Long, T.D., Cathers, T.A., et al. Treatment of Painful Skin Ulcers with Topical Opioids. Journal of Pain and Symptom Management 17:288-92, 1999.
27. Ballas, S.K. Treatment of Painful Sickle Cell Ulcers with Topical Opioids. Blood 99:1096, 2002.
28. Berg, J.O., Mossner, B.K., Skov, M.N., et al. “Antibacterial Properties of EMLA and Lidocaine in Wound Tissue Biopsies for Culturing.” Wound Repair and Regeneration 14:581-5, 2006.
29. Minniti, C.P., Delaney, K.M., Glorbach, A.M., et al. Vasculopathy, Inflammation, and Blood Flow in Leg Ulcers of Patients with Sickle Cell Anemia. American Journal of Hematology 89:1-6, 2014. doi: 10.1002/ajh.23571.
30. Winter, G.D. “Formation of the Scab and the Rate of Epithelialization of Superficial Wounds in the Skin of Young Domestic Pigs.” Nature 193:293-4, 1962.
31. Ovington, L.G. “Hanging Wet-to-Dry Dressings Out to Dry.” Home Healthcare Nurse 19:477-83, 2001.

32. O'Meara, S.M., Cullen, N.A., Majid, M., et al. "Systemic Reviews of Wound Care Management: (3) Antimicrobial Agents for Chronic Wounds; (4) Diabetic Foot Ulceration." Health Technology Assessment 4:1-238, 2000.
33. National Institute for Clinical Excellence. Type 2 Diabetes. Prevention and Management of Foot Problems: Clinical Guideline 10. London, UK: National Institute for Clinical Excellence, 2004.
34. Leaper, D.J. "Silver Dressings: Their Role in Wound Management." International Wound Journal 3:282-94, 2006.
35. Alvarez, O.M., Mertz, P.M., Eaglstein, W.H. "The Effect of Occlusive Dressings on Collagen Synthesis and Re-epithelialization in Superficial Wounds." Journal of Surgical Research 35:142-8, 1983.
36. Lamme, E.N., Gustafsson, T.O., Middelkoop, E. "Cadexomer-Iodine Ointment Shows Stimulation of Epidermal Regeneration in Experimental Full-Thickness Wounds." Archives of Dermatological Research 290:18-24, 1998.
37. Holloway, G.A. Jr, Johansen, K.H., Barnes, R.W., et al. "Multicenter Trial of Cadexomer Iodine to Treat Venous Stasis Ulcer." Western Journal of Medicine 151:35-8, 1989.
38. Treadwell, T.A. Basics of Wound Care. Grand Rounds Lecture presented at Hopital de L'Universite D'Etat D'Haiti, Port-au-Prince, Haiti, July 2, 2014.
39. Serjeant, G.R., Gallaway, R.E., Gueri, M.C. "Oral Zinc Sulphate in Sickle Cell Ulcers." Lancet 2:891-2, 1970.
40. Treadwell, T., Fowler, E., Jensen, B.B. "Management of Edema in Wound Care: A Collaborative Practice Manual for Health Professionals." In C. Sussman, B.B. Jensen, eds. Wound Care: A Collaborative Practice Manual, 4th ed. New York, NY: Lippincott Wilkins & Williams, 2012.
41. Gordon, S., Bui, A. "Human Skin Equivalent in the Treatment of Chronic Leg Ulcers in Sickle Cell Disease Patients." Journal of the American Podiatric Medical Association 93(3):240-1, May-June 2003.
42. Schultz, G.S., Falanga, V., et al. "Wound Bed Preparation: A Systematic Approach to Wound Management." Wound Repair and Regeneration 11(Suppl):1-28, 2003.
43. Treadwell, T.A., Fuentes, M.L., Walker, D. "Wound Bed Preparation Prior to the Use of Bi-layered Tissue Engineered Skin: The Role of Protease Modulation." Wound Repair and Regeneration 16:A19, 2008.
44. Chin, G.A., Schultz, G.S. "Treatment of Chronic Ulcer in Diabetic Patients with a Topical Metalloproteinase Inhibitor, Doxycycline." Wounds 15(10):315-23, 2003.
45. Cullen, B., et al. "Mechanism of Action of Promogran, a Protease-Modulating Matrix, for the Treatment of Diabetic Foot Ulcers." Wound Repair and Regeneration 10:16-25, 2002.
46. Eckman, J., Platt, A. "Leg Ulcers." Sickle Cell Information Center Guidelines. Available at: www.scinfo.org/legulcr.htm. Last accessed July 1, 2014.

47. Heckler, F.R., Dibbell, D.G., McCraw, J.B. “Successful Use of Muscle Flaps or Myocutaneous Flaps in Patients with Sickle Cell Disease.” Plastic and Reconstructive Surgery 59:902-8, 1997.
48. Khouri, R.K., Upton, J. “Bilateral Lower Limb Salvage with Free Flaps in a Patient with Sickle Cell Ulcers.” Annals of Plastic Surgery 27:574-6, 1991.
49. Richards, R.S., Bowen, C.V.A., Glynn, M.F.X. “Microsurgical Free Flap Transfer in Sickle Cell Disease.” Annals of Plastic Surgery 29:278-81, 1992.
50. Best, J.P., Daoud, M.S., Pittelkow, M.R., et al. “Hydroxyurea-Induced Leg Ulceration in 14 Patients.” Annals of Internal Medicine 128:29-32, 1998.
51. Weinlich, G., Schuler, G., Greil, R., et al. “Leg Ulcers Associated with Long-Term Hydroxyurea Therapy.” Journal of the American Academy of Dermatology 39:372-4, 1998.
52. Lopes, F.C., Ferreira, R., Albuquerque, D.M., et al. In Vitro and In Vivo Anti-Angiogenic Effects of Hydroxyurea. Mircovascular Research 94:106-13, 2014.
53. Flores, F., Eaglstein, W.A., Kirsner, R.S. “Hydroxyurea-Induced Leg Ulcers Treated with Apligraf.” Annals of Internal Medicine 132(5):417-8, March 2000.
54. Steinberg, M.H. “Management of Sickle Cell Disease.” New England Journal of Medicine 340:1021-30, 1999.
55. Sher, G.D., Olivieri, N.F. “Rapid Healing of Chronic Leg Ulcers During Arginine Butyrate Therapy in Patients with Sickle Cell Disease and Thalassemia.” Blood 84:2378-80, 1994.
56. Atweh, G.F., Sutton, M., Nassif, I., et al. “Sustained Induction of Fetal Hemoglobin by Pulse Butyrate Therapy in Sickle Cell Disease.” Blood 93:1790-7, 1999.
57. McMahon, L., Tamry, H., Askin, M., et al. Randomized Phase II Trial of Arginine Butyrate with Standard Local Therapy in Refractory Sickle Cell Leg Ulcers. British Journal of Haematology 151:516-24, 2010.
58. Mery, L., Girot, R., Aractingi, S. “Topical Effectiveness of Molgramostim (GM-CSF) in Sickle Cell Leg Ulcers.” Dermatology 208:135-7, 2004.
59. DeFranceschi, L. “Pathophysiology of Sickle Cell Disease and New Drugs for the Treatment.” Mediterranean Journal of Hematology and Infectious Diseases 1(1): e2009024, 2009. Available at: http://www.mjhid.org/article/view/5239. Last accessed July 1, 2014.
60. Lopez, B.L., Kreshank, A.A., Morris, C.R., et al. “L-Arginine Levels Are Diminished in Patients with Sickle Cell Crisis in the Emergency Department.” British Journal of Haematology 120:532-4, 2003.
61. Boykin, J.V., Baylis, C. “Homocysteine—A Stealth Mediator of Impaired Wound Healing: A Preliminary Study.” Wounds 18(4):101-4, 2006.
62. Treadwell, T., Walker, D., Mara, L. The Treatment of Sickle Cell Ulcers with the Combination Therapy of L-Methylfolate, Pyridoxal 5′-phosphate, and Methylcobalamin. Poster Presentation, SAWC, Las Vegas, NV, October 2011.

63. Treadwell, T., Walker, D., Nicholson, B.J., et al. The Use of Nitric Oxide/Plasma Therapy in the Treatment of Recalcitrant Wounds. Poster Presentation, Spring SAWC, Orlando, FL, April 2014.
64. Chang, J., Shi, P.A., Chiang, E.Y., et al. Intravenous Immunoglobulins Reverse Acute Vasooccluion Crises in Sickle Cell Mice Through Rapid Inhibition of Neutrophil Adhesion. Blood 11(2):915-23, 2008.
65. Hayek, S., Dibo, S., Baroud, J., et al Refractory Sickle Cell Ulcer: Is Heparan Sulphate a New Hope? International Wound Journal 13(1):35-8, 2014. doi: 10.111/iwj.12217.
66. Bernaudin, F., Socie, G., Kuentz, M., et al. SFGM-TC. Long Term Results of Related Myeloablative Stem-Cell Transplantation to Cure Sickle Cell Disease. Blood 110:2749-56, 2007.
67. Bernaudin, F., Pondarré, C., Galambrun, C., et al. Allogeneic/Matched Related Transplantation for β-Thalassemia and Sickle Cell Anemia. Advances in Experimental Medicine and Biology 1013:89-122, 2017.
68. Connor, J.L., Minniti, C.P., Tisdale, J.F., et al. Sickle Cell Anemia and Comorbid Leg Ulcer Treated with Curative Peripheral Blood Stem Cell Transplantation. The International Journal of Lower Extremity Wounds 16(1):56-9, 2017.
69. Rubin, R. Gene Therapy for Sickle Cell Disease Shows Promise. In Health Agencies Update. JAMA 321(4):334, 2019.

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Surgical Wounds and Surgical Reconstruction

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Objectives

After completing this chapter, you will be able to:

- state the normal trajectory of surgical wound healing
- describe the appropriate suture line care to promote optimal healing
- identify the strategies that can decrease the occurrence of a surgical site infection (SSI)
- explain the intrinsic and extrinsic causes of surgical wounds that have failed to heal
- define the reconstructive ladder
- describe how the reconstructive ladder is used to achieve wound closure
- explain the surgical principles that guide care of patients who require skin, tissue, or bone transplantation for wound closure.

Introduction

Surgical wounds are common in hospitalized patients; however, due to the body's amazing ability to heal acute wounds, hospitalized patients are seldom seen by wound care specialists unless their wounds have failed to heal. This chapter addresses normal surgical wound healing, appropriate suture line care, strategies to prevent a surgical site infection (SSI) from occurring, common surgical wounds, complex wounds healed via surgical procedures, and surgical wounds that have failed to heal in a timely manner.

Surgical Wounds

To better understand the ideal surgical wound healing outcome, it is important to appreciate the concepts and normal trajectory of wound healing.[1] There are three overarching phases of wound healing: the inflammatory phase, the proliferative phase, and the remodeling phase.[2,3] Within these overarching phases are four physiological events: hemostasis, inflammation, repair, and remodeling.[3,4] An acute wound will heal through these physiological processes in an orderly and sometimes overlapping fashion. It takes around 21 to 30 days for an acute wound to complete the inflammatory and proliferative phases.[3] The final phase, remodeling, can take from 1 to 2 years to complete.[2,5] Partial-thickness and full-thickness wounds pass through these four physiological events. The only exception is a partial-thickness wound that only involves the epidermis. When only the epidermal level requires healing, it heals through a regeneration process.[6,7] There are three possible wound healing scenarios or responses. A wound can heal through primary intention such as through the surgical approximation of edges. When a wound heals without surgical intervention but by granulation and epithelization, it is termed secondary intention. If a wound has remained opened for a period of time and then heals through delayed surgical closure (e.g., flap, graft, primary closure) or by using an advanced modality, it is considered healing through tertiary intention.[2,8]

The most common surgical wound is the suture line, which heals through primary intention. Any dressing that is applied over the suture line should have the ability to protect the wound.[9,10] The suture line should be protected for at least 48 hours until the natural skin barrier is typically formed.[2,11–15] Thus, the intent of the primary surgical dressing is to act as a “physical barrier” protecting the surgical site from external trauma and bacterial contamination.[1,12] The primary dressing should be kept clean and dry and only changed when saturated. Saturated dressings can encourage bacterial contamination.[1,12] The more traditional surgical dressing is gauze and tape or gauze with a transparent dressing. More recently, however,

some surgeons have been opting to use advanced composite dressings that may or may not offer antimicrobial action.[1] The reasons for avoiding gauze in favor of a composite dressing may be due to gauze's properties and/or the materials used to hold it in place. Gauze is known to have limited absorbency capacity[16] and can harm newly formed tissue upon removal and increase the patient's pain if there is dried, drainage residue present,[16–18] and the traditional tapes (nonsilicone) and transparent films used to keep the gauze in place may cause skin stripping and/or moisture-associated skin damage (MASD).[1,19]

Promoting surgical site healing and the avoidance of an SSI require evidence-based measures that become inherent in daily practice. In 2008, when the Centers for Medicare and Medicaid Services (CMS) put forth the determination that hospital-acquired conditions (HACs) would no longer receive additional payment, SSIs were one of the first eight conditions.[20] SSIs are a global problem and still growing. In the United States alone, up to 300,000 SSIs occur each year and are considered a costly and common healthcare-associated infection (HAI).[21–23] Equally alarming is the percentage of SSIs (13.3% to 21.8%) that are detected and managed post discharge versus those that may not be detected due to poor surveillance.[21,24–27] SSI percentage has been reported per institution, type of surgery, and type of SSI and by phase of occurrence (e.g., hospital, readmission, postdischarge).[9,25,26,28,29] The occurrence of an SSI can increase length of stay (LOS),[21] readmission rates,[25] morbidity,[1] and mortality.[1,21,22]

There are three types of SSIs. The first type is superficial incisional SSI involving the skin only or the subcutaneous tissue of the incision. The second type is deep incisional SSIs involving the fascia and/or muscular layers. Deep incisional SSIs are then subdivided into deep incision primary and deep incision secondary. Deep incision primary SSI is identified as the primary incision in a patient who had more than one incision created during one operation. Deep incision secondary SSI is identified as the secondary incision in a patient who had more than one incision created during one operation. The last type is organ/space that involves any part of the body that has been

opened or manipulated during the procedure except the skin incision, fascia, or muscle layers.[21] Aside from costs that can be accrued from an SSI occurrence, additional costs can be accrued when an SSI results in an acute wound that then advances to a chronic wound.[1] Undoubtedly, patients with a higher comorbidity index can be more susceptible to an SSI, which can be the same factors that delay wound healing.[1]

Due to this crisis, the literature has been growing regarding practice recommendations to avoid SSI occurrence and improve patient outcomes.[1,9,21,30,31] There are several global organizations and societies that have put forth best practice recommendations such as the World Health Organization (WHO), the National Institute for Clinical Excellence (NICE) in the United Kingdom, the American College of Surgeons and Surgical Infection Society, the Centers for Disease Control and Prevention (CDC), and a larger group of societies and organizations spearheaded by the Society for Healthcare Epidemiology of America (SHEA).[9,21,27,30,31] Essentially, the avoidance of an SSI can be dependent on optimizing or modifying intrinsic and extrinsic factors well before the surgery occurs with best practice efforts remaining in place even after the patient has been discharged from the facility.[1] While there are numerous best practice recommendations, some examples include (a) addressing patient conditions before surgery such as uncontrolled hyperglycemia and smoking; (b) using clippers rather than razors, if necessary; (c) timing of antibiotic administration before the first incision is made; (d) maintaining normothermia during the procedure; (e) using proper skin closure technique; and (f) caring of postoperative suture line care.[9,21,27,30,31] The obvious emphasis is that SSIs can be preventable when evidence-based guidelines are put into practice via a multifaceted and teamwork approach.[1,21,22,25,26,28,29] A surgical wound's speed of healing and the avoidance of an SSI are determined by the care taken with surgical preparation occurring well before the patient's admission and continuing well after surgery.[1,25]

Extrinsic and Intrinsic Causes of Nonhealing Wounds

Causes of nonhealing wounds can be grouped into two large categories: extrinsic and intrinsic. Extrinsic causes are those factors that exist outside of the wound itself, such as pressure, ischemia, smoking, and malnutrition. Intrinsic factors exist within the wound, such as infection, tension, and arterial insufficiency. Determining which of these factors is present in a wound is an important initial step in wound care. Removing any obstacles to healing is also important; local treatments won't be effective in healing the wound if the underlying cause of the disruption in healing is not addressed.

Wound care focuses on removing the obstacles to healing. Although wound care has seen technological advances, the great 16th-century surgeon, Ambroise Paré, is still correct in that we as healthcare providers don't heal wounds, we create them, and nature heals them.[32] Indeed, our role is to support the body so that the wound can heal. Wound care practitioners must exercise caution when moving too quickly into treatments for wounds until the underlying causes of the delay in wound healing are fully understood.

Wound History

There are four major questions that need to be addressed by the clinician:

1. How and when did the wound occur?
2. When did the wound's healing trajectory change from normal to abnormal and why?
3. What known factors are obstructing the wound from healing?
4. What therapies have been attempted?

When examining a patient with a nonhealing surgical wound, it's imperative that the initial reason for surgery and the operation performed be fully understood. The answers to the following questions must be clear in the examiner's mind: what was the preoperative condition that required surgery? For example, did the patient have gastric bypass or repair of ruptured abdominal viscera? What was the patient's condition before surgery? Was the patient healthy or was the patient chronically ill due to diabetes or cancer? What was the intended operation? Were organs or tissues removed? Was the operation clean or contaminated (e.g., penetrating trauma, perforated diverticulum)? Were foreign materials implanted? Was the operation carried out as planned? For example, has the wound been closed with permanent sutures or mesh? Has healing been occurring along a normal trajectory until now? If so, what has happened to alter the course of healing? What medications is the patient taking that might impact healing? Has the nutritional status of the patient been normal since surgery? Was it normal prior to surgery?

From this initial assessment, a comprehensive understanding of the patient who has the wound, not just the wound itself, will be obtained. And, although treating the wound is of utmost importance, treating the patient, whose body will heal the wound, is equally imperative.

Goals of Care

For acute wounds, wound closure with the return of form and function is the overarching goal of care. Left alone to close via contraction and scar formation, wounds seldom have either form or function and will often recur, look unpleasant, and lead to pain because the scar does not stretch with movement. The ability to reach ideal and complete healing without scarring, such as seen in fetal wounds, is currently impossible. All full-thickness skin injuries heal with a scar. Therefore, the return of acceptable healing with sustained function and anatomic continuity becomes the ideal end point. The surgeon is constantly mindful of the consequences to leaving the wound open, balanced against the consequences of surgical closure and undergoing anesthesia.

Reconstructive Ladder and Planning Reconstruction

A decision-making process in choosing the appropriate method to achieve wound closure is based on the following information and the "rungs" of the reconstructive ladder (Fig. 20-1). Following are some important questions to consider.

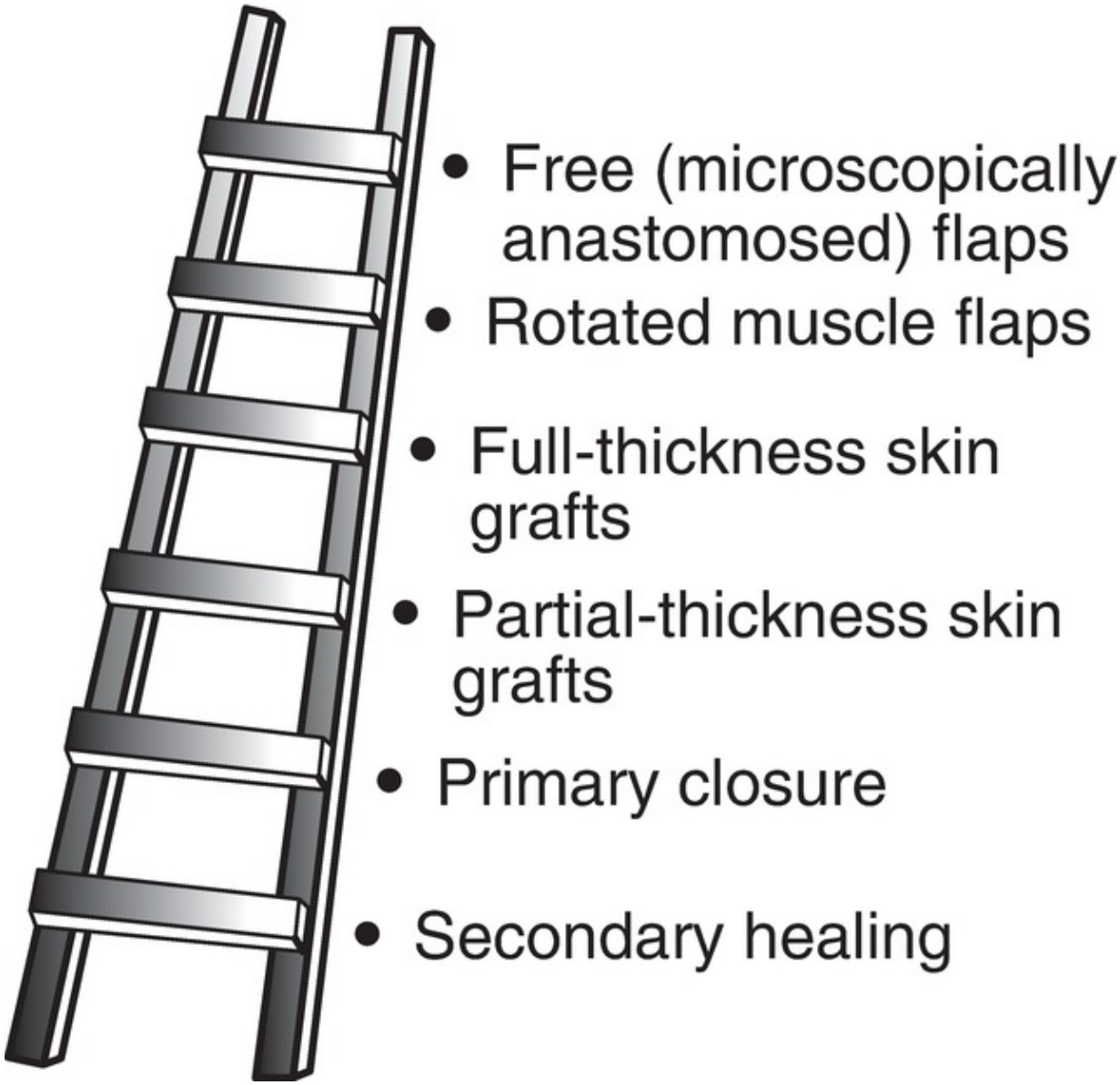

Figure 20-1. Reconstructive ladder. The reconstructive ladder is used to determine the method to replace missing tissue from a wound bed. The ladder shows the simplest method—simply allowing the wound to heal on its own—at the bottom of the ladder.

Is the Wound Missing Tissue?

If the wound hasn't lost any tissue, it may be possible to close it primarily. Wounds are capable of healing by primary intention, which occurs when the wound edges are approximated (pulled together) and retained by sutures, staples, or glue. The dynamics of healing begin, and new tissue is synthesized. A healing ridge (an induration beneath the skin, extending to approximately 1 cm) forms directly under the suture line between 5 and 9 days after surgery. All forms of wound healing lead to scar, and primary wound closure is no different. If the scar is under tension, it is prone to exaggerated scar formation, such as hypertrophic scar.

What Kind of Tissue, If Any, Is Missing?

Wounds that only lack portions of the skin may be allowed to granulate closed if they're small, or the skin may be grafted to speed the healing process. Wounds that have damaged tendon, muscle, or bone may require transplantation of this tissue to provide form and function (Fig. 20-2). Such operations require flaps of the skin, muscle, fascia, or bone. Such flaps are named for their composition, such as an osteocutaneous flap, which is a bone and skin flap.

Figure 20-2. Right foot surgical transplantation/repair of tendon. (Photo courtesy of S. Baranoski.)

Flaps (as opposed to grafts) bring their blood supply with them and either are locally transported to the wound or are from a distant site (free flap). The free flap employs the surgical technique of freely removing tissue from one area, detaching the nutrient artery and vein from their supply vessels, and moving the tissue to the recipient site where these vessels are reattached to new supply vessels. For example, a free radial forearm flap contains radius bone, overlying muscle, and skin. It is commonly used to reconstruct the face and jaw after wide excision of cancer of the mandible and floor of the mouth.

What Donor Site Morbidity May Occur?

Donor sites have some form of scarring and may exhibit some loss of function, depending on the tissue removed. Split skin graft donor sites should heal with minimal effort; there is no loss of function but a scar remains. When the breast is reconstructed using the latissimus dorsi muscle from the back, the woman may lose some functions of the shoulder. Although some patients perceive the loss of function as tolerable, other patients—for example, a tennis player—may undoubtedly perceive it differently. If a thumb is lost, a patient may opt to transplant the great toe to provide opposition for hand function; however, most people wouldn't want to sacrifice a thumb to replace the great toe. Some cultures (e.g., Japan) place great importance on the toes and prefer to use the second toe rather than the great toe for thumb reconstruction. In other words, the degree of loss a patient is willing to experience is proportional to his or her need for the sacrificed tissue.

What's the Simplest Method to Achieve Wound Closure?

The easiest method available to close the wound is often used first. Wounds that can heal quickly on their own via granulation and epithelialization are allowed to do so. However, when large wounds or

wounds over thin skin (e.g., sacrum) are allowed to granulate closed, the resultant scar is often unstable and prone to injury and ulceration. Skin grafting is the next simplest method and is used to treat wounds that are missing only the skin. Skin grafting can be full or partial thickness, depending on the kinds of tissue missing in the recipient site and the condition of the site. Grafting of any tissue type relies on that tissue to revascularize at its recipient site. Wounds that could close on their own, but with accompanying contracture and loss of function, may also be grafted. If the muscle is missing, muscle flaps may be used to fill the wound defect or cavity, but the muscle isn't typically made functional (i.e., an insertion, origin, and nerve aren't restored to create a functional muscle). One notable exception is the gracilis free flap to restore the function of the zygomaticus major after facial nerve injury. The blood supply to the muscle is restored so that it remains viable. The ample blood flow into the muscle is commonly used to treat complex wound problems such as osteomyelitis. Restored blood flow may also transport antibiotics and immune cells to the wound.

Muscle has been shown to supply overlying islands of skin through a series of vessels that perforate the muscle body. Surgeons can simultaneously transplant a muscle and the island of skin to both fill a wound cavity and provide skin coverage. Such a flap is called a musculocutaneous flap. These flaps are typically named after the muscle, as in the case of the tensor fascia lata flap, which is used to close a trochanteric pressure ulcer. If a large amount of soft tissue is missing, the muscle may be transplanted and then covered with skin grafting to achieve the same effect. Such an operation is called a muscle flap and split-thickness skin graft, with the specific muscle being grafted added to the name.

Muscle is brought to the recipient site in one of two ways:

- It's rotated along the arc of its original blood supply left intact.
- It's freed from its blood supply and the artery and vein are reattached via a microscope at the recipient site.

Freeing the muscle, skin, and other parts of a flap allows the flap to be used for reconstruction in areas that the muscle normally couldn't reach, such as the lower third of the leg. It's important to note that, as

often as possible, tissue should be replaced with similar tissue. Similar hair bearing, appearance, and thickness improve aesthetic appearance of the reconstructed wound.

Overall, this escalation in complexity of reconstruction is referred to as the “reconstructive ladder” and is the cornerstone of reconstructive plastic surgery.

Wounds Without Missing Tissue

Some wounds don't require grafts because they aren't missing tissue, or local tissue can be undermined and recruited to close the original wound.

Simple Lacerations

Traumatic wounds are often missing little or no tissue and can be closed primarily; however, the full extent of the injury must be known before any closure is attempted. Laceration of arteries and veins is usually obvious by the amount of bleeding present. Facial lacerations are especially bloody due to the robust blood supply in the face. The patient with a facial laceration is assessed for function of motor and sensory nerves, muscle, and parotid and other salivary glands in the area prior to systemic or local anesthesia, which would obscure the findings. Repair of vessels, tendons, ligaments, and nerves is commonly completed in the operating room because it is a sterile environment and sometimes has the added benefit of an operating microscope. Wounds are irrigated copiously to remove any debris. Skin closure is accomplished by undermining surrounding tissue to facilitate its movement and suturing layers of tissue with minimal tension. Drains may be placed in wounds with contamination or large amounts of dead space. The appearance of the wound should ideally mimic the uninjured appearance. However, traumatic amputation or extensive debridement may leave an appearance that's less than acceptable.

Removing all forms of tension on the wound reduces the scar. Wounds that must be mobilized to gain function, such as incisions over joints, heal with wider scars, and this may be disturbing to the patient's self-image. Stented dressings, immobility (e.g., limited chewing or talking with facial lacerations), and thin applications of topical antibiotics may help to minimize scars. Moist wound healing techniques, used throughout the healing process, will minimize healing time and potentially the amount of scarring. However, it's important for the patient, the patient's family, and members of the healthcare team to realize that scarring is inevitable and only after the

scar has matured, which can take more than 1 year, will scar revision be attempted. Silicone-based dressings may be used over some healed wounds to help minimize scar buildup during the maturation phase. This has only been shown helpful with hypertrophic scarring. The reader is referred to an excellent evidence-based review of emergency wound management.[33]

Extensive Lacerations

Extensive lacerations, while unsightly, are seldom life-threatening. Lifesaving care of the heart, lungs, and brain precedes definitive wound care. Initial wound care includes debridement of obvious dirt, glass, grass, or other foreign bodies. Massive lacerations are packed with moist dressings to prevent tissue desiccation, and, when the patient is stable, the wounds are debrided and surgically closed, if possible. Extensive wounds may require multiple debridements until viable tissue is present; these wounds may also require more complex forms of delayed closure, such as flaps.

Wounds that are not closed within the first 6 hours after injury are considered contaminated and are at infectious risk if primarily closed after that time. Some clean facial wounds are an exception to this rule, and their closure may be delayed longer in healthy patients who are on systemic antibiotics.

Penetrating Abdominal Wounds

Penetrating abdominal wounds include stab wounds, impalements, gun shots, and so forth. These are serious wounds due to direct tissue injury, risk of contamination and sepsis, and the potential for excessive bleeding. Depending on the degree of contamination from environmental agents (e.g., dirt) or penetration of the bowel or bladder, the wound may not be closed completely. Frequently in traumatic injuries, these wounds are not closed external to the fascia. If the fascia cannot be closed, leaving the abdomen open is also a challenge. These patients require multiple re-explorations while simultaneously reducing or controlling abdominal fluid secretion and preserving the fascia for closure. Currently, no standard protocols exist for the management of the open abdomen, and care is based on

clinical judgment by surgeon experience. Most often, individual major trauma centers have their own experience and evidence-based protocols.

Abdominal Compartment Syndrome

Abdominal compartment syndrome (ACS) is similar to compartment syndrome of the extremity. ACS can occur during elective abdominal operations, following abdominal trauma or pelvic fracture, and with pancreatitis. Normal intra-abdominal pressure in surgical patients is 2 to 10 mm Hg; a value above 12 mm Hg indicates intra-abdominal hypertension, and a value above 20 mm Hg is considered an indicator of ACS. Intra-abdominal pressure can be determined using simple water column manometry with a bladder catheter. When pressure is mildly increased to 10 to 15 mm Hg, the cardiac index rises due to compression of the vena cava. With further elevations, there is progressive organ dysfunction due to intra-abdominal pressure on the abdominal organs and vena cava. Direct compression on the hollow intestine and portacaval system causes these structures to collapse. When the bowel is compressed, it becomes ischemic, allowing bacteria to thrive. Vasoactive substances such as histamine and serotonin increase endothelial permeability, further capillary leakage impairs red blood cell transport, and ischemia worsens. As pressure rises, ACS not only impairs visceral organs but also damages the cardiovascular and the pulmonary systems; it may also cause a decrease in cerebral perfusion pressure. Therefore, ACS should be recognized as a possible cause of decompensation in any critically injured patient.[34,35]

It is important to include ACS in a wound healing chapter because the treatment of ACS is not closure of the abdomen. If the abdomen is at all difficult to close, this procedure should be abandoned and alternative techniques applied. A good rule of thumb is as follows: when looking at the abdomen horizontally, if you can see the gut above the level of the wound, consider leaving the abdomen open and using temporary closure.

The easiest way to control the open abdomen is to use a silo-bag closure. This short-term therapy has increasingly been replaced by

negative pressure wound therapy (NPWT). The use of NPWT has been shown to allow successful fascial closure in patients who in the past would have required mesh grafting for closure. While NPWT increases the use of primary closure, it also shortens time to recovery.[36,37]

Abdominal Wound Dehiscence and Evisceration

Wound dehiscence is the separation of the edges of a surgical wound. The strength of a wound lies in the musculoaponeurotic layer of the abdomen. In the early postoperative period, wounds stay closed as a result of the strength of the sutures used or through normal healing processes as muscles regain their strength. However, some wounds are susceptible to dehiscence, thus requiring tissue transplantation.

Risk factors for wound dehiscence may be technical or related to patient factors. Technical factors are due to the type of closure used. Indeed, wound dehiscence may occur because sutures break, sutures stretch or cut through the tissue, knots slip, the suture is too thin, or an insufficient number of sutures are used. Closure is best achieved when long-lasting, absorbable, or permanent sutures are used, with secure knots that do not slip or invite bacteria to harbor in them. Sutures should be placed about 1 cm from the abdominal wound and 1 cm apart from each other. This closure places the suture in healthy fascia that will not be cut by suture material. In wounds that are healing securely, a healing ridge of palpable thick tissue about 0.5 cm appears along the incision. This ridge is almost always absent in wounds that rupture.

Risk factors for abdominal wound dehiscence were reported in a large retrospective study.[38] The most significant patient-related factors for dehiscence were age over 50 years, male gender, emergency operation, longer operative times, postoperative coughing, hypertension, steroid use, cancers, ascites, chronic pulmonary disease, jaundice, anemia, and wound infection. A risk score was computed with the highest risk factors being age over 70 years; ascites; vascular surgery; surgery on the esophagus, stomach, or large bowel; coughing; and wound infection.[38]

Obesity, heavy coughing or retching, and ascites, all of which strain the wound during healing, predispose to dehiscence. However, many surgeons believe that if a wound is closed securely, these complications will not occur. Usually, the first sign of an impending problem is the sudden discharge of serosanguineous fluid from the wound, but some patients present with sudden evisceration following an episode of coughing or retching. When the edges of the wound separate and internal organs such as the intestines are protruding from the wound, this is known as evisceration.

Evisceration is a frightening experience for the patient. Immediate treatment includes helping the patient to remain calm, medicating for pain, and holding the abdominal contents in the abdomen by keeping the viscera moist with a sterile moist towel. Exposed intestines should not be forced back into the abdomen. The patient should be kept NPO and prepared for immediate surgery. Lower the head of the bed so it is flat or no more than 20 degrees. Monitor the patient's vital signs and assess for signs and symptoms of shock.[39] The wound is explored urgently in surgery, devitalized tissue is excised, and the abdomen is closed with nonabsorbable interrupted suture taking secure bites into healthy tissue. Hernia formation is relatively common, reaching 30% in wounds that eviscerated and reclosed. If the wound has dehisced and the patient cannot tolerate another anesthetic, the wound can be packed with dressings, an abdominal binder used, or a vacuum-assisted closure applied to obtain secondary healing. These wounds will inevitably develop a hernia.

Wounds Requiring Tissue Transplantation for Closure

Tissue transplantation is a phrase used to describe closure techniques for a group of wounds that have missing tissue. In order to achieve closure, various tissues (skin, muscle, fascia, bone) can be grafted or flapped into the wound. These wounds can be quite complex to manage.

Burns

Surgical management of burn wounds includes escharotomy, excision of eschar, skin grafting, and scar revision. Because many burned patients must undergo multiple operations, it is beneficial to have a dedicated team for these patients to reduce some of the anxiety associated with surgery, anesthesia, and pain management. It is wise to have severely burned patients go directly from their inpatient room to surgery, not stopping in preoperative areas.

Escharotomy

Escharotomy is a decompressive operation to reduce compartment syndrome in the extremities, respiratory compromise, ACS, and even orbital compression. Full-thickness burned skin cannot stretch; therefore, during fluid resuscitation, the fluid that moves into the interstitial spaces places pressure on vessels and nerves. Indications for escharotomy include compartment syndrome of the extremities (pallor, pain, pulselessness, paresthesia, and paralysis), falling oximetry with respiratory difficulty, increased abdominal pressure, and changes in vision (with increased periorbital pressures). Escharotomy is completed by incising the inelastic burned tissue along the longitudinal axis of the extremities or across the chest and/or abdomen. The surgical site is left open to allow for both edema and chest wall movement with respiration. Once the capillary bed has stabilized and fluid leaves the interstitial spaces, the escharotomy incisions may approximate. The incisions are not closed, but rather are allowed to heal secondarily or may be grafted.

Excision of Eschar

Years ago, eschar was removed slowly with weeks of daily submersion in tanks and debridement with scissors. This process is still used for some burns, but many burns today are debrided with pressurized water knives, and some burns are completely excised and grafted early to promote rehabilitation. Burns of the hand are typically managed in this fashion in order to quickly mobilize the hand and restore function.

Skin Grafting

The most accepted way to permanently heal a full-thickness burn wound is to skin graft the wound. Skin grafts are portions of the patient's skin removed from a donor site of unburned skin or healed burned skin that are moved to cover an open burn wound. They are often referred to as split-thickness skin grafts. Skin grafts are harvested in the operating room and placed on the freshly debrided burn wound. The skin graft lives off of the serum in the wound bed; it does not have a blood supply (unlike a flap). As the capillary beds in the wound bed grow into the skin graft, it becomes pink in color. This pink color is called a "take" and often expressed as a percentage (e.g., 50% take of the split-thickness skin graft).

A crucial aspect of skin graft healing is immobilization of the graft onto the wound bed. Most surgeons use fibrin glue, splint the graft in place with a bolster, or suture it to the edges of the wound. Because edema in the space between the wound bed and the skin graft can separate the graft from capillary ingrowth, NPWT is often used to promote attachment. If NPWT is used, the suction should be continuous and not interrupted.

The dressings are usually removed at 72 hours to inspect the graft. Dressings after that point vary, but many surgeons continue to dress the newly grafted site with an antimicrobial dressing.

Skin grafts fail for several reasons. The primary reason is edema in the wound bed that lifts the grafted skin off the bed. It is imperative that all precautions to keep the grafted area elevated be followed. Too many well-meaning patients have convinced nurses that during a quick trip to the bathroom, rather than the bedside commode, the leg

will remain elevated. Unfortunately, those short periods of time with the leg down may be all that is required to allow edema to lift the graft off of the wound bed!

Patient Teaching

Instruct patients to use the commode rather than the bathroom after a skin graft to the leg.

Skin grafts can also fail due to infection in the wound. If infection is suspected, it should be ruled out with quantitative cultures of the wound bed prior to grafting.

Pruritus is a common and distressing problem in healed burn wounds. Skin grafts do not carry sweat or oil glands with them, making the transplanted skin prone to dryness and pruritus from heat retention. In addition, burn scars are not elastic, making them prone to injury. Persistent pruritus is estimated to occur in about 87% of burned patients.[40] Methods to treat pruritus have not been compared systematically, and the use of histamine receptor agonists is commonly based on the belief that pruritus is mediated by mast cells. Newer thoughts on the etiology of pruritus following skin grafting are that it is a variant of pain, and newer treatments for pruritus have examined eutectic mixture of local anesthetic (EMLA) cream, transcutaneous electrical nerve stimulation (TENS), colloidal oatmeal, massage, and sedatives. The problem of pruritus is in great need of ongoing study.

Scar Revision

Scars continue to mature over 6 to 12 months following a burn. Burned areas that were allowed to heal without grafting have the most robust scars. When the scars reach across joints, the joints can be pulled into contracture. Z-plasty is a procedure that can be performed

if there is adequate tissue in one dimension and not the other, for example, adequate tissue in length but not width. Z-plasty transfers the excess tissue to the area in need of tissue, thereby releasing the contracture. Many times, however, there is not enough tissue in either direction, and so skin grafting is needed.

Nonhealing Surgical Wounds

Surgical wounds can be slow to heal because of underlying disease states, such as infection, poorly controlled diabetes, impaired arterial blood flow, protein–calorie malnutrition, and compromised immunity (Fig. 20-3). By convention, any surgical wound of 3 to 4 weeks' duration that is not responding to conventional therapy is considered nonhealing. From the surgical perspective, only debridement can be offered as initial treatment.

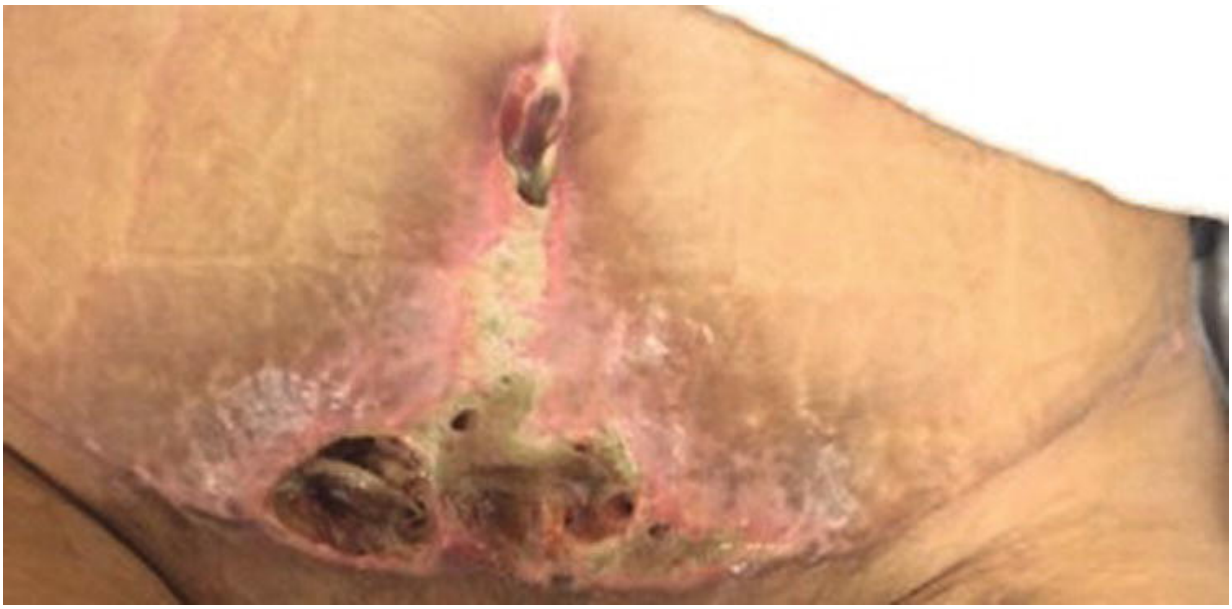

Figure 20-3. Full-thickness skin loss abdominoplasty. (Photo courtesy of Barbara Delmore.)

Infection, including the development of biofilms, is a common culprit of nonhealing. Biofilms promote inflammation, and inflammation delays healing. Common bacterial causes include *Staphylococcus*,

Pseudomonas, and *Enterobacter*. Debridement, performed either traditionally or using low-frequency ultrasound, destroys the biofilm and is an important component of wound care.[41]

Median Sternotomy Wounds

Median sternotomy incisions are extremely complex wounds to close following surgery. Bone stabilization is usually done by rigid fixation and immobilization, but the sternum is often closed with wire and subjected to constant movement with respiration.

Infected medial sternotomy wounds following cardiac surgery are a dreaded complication. High-risk patients include those who are obese (body mass index over 30) and those with diabetes, heart failure, previous myocardial infarction, urgent operative status, hypertension, and/or large breasts. Perfusion time over 200 minutes, use of an intra-aortic balloon pump, and three or more distal anastomoses are risk factors for very serious infections, but these conditions rarely occur.[42,43] Control of blood glucose and improvement of heart failure prior to surgery are helpful interventions.

Early wound infection, such as the aforementioned suppurative mediastinitis, appears as cellulitis, purulent wound drainage, and obvious tracking between the skin, sternum, and mediastinum. Left untreated, these infections smolder down into the mediastinum and may even extend into aortic suture lines, prosthetic grafts, and intracardiac prostheses. Local wound care is based on the condition of the wound and the condition of the patient. Wound bed preparation may include packing, debridement, or the use of NPWT, which may be used as a first line of treatment because it splints the chest wall. NPWT can be used as a bridge to allow the patient to recover or stabilize. A systematic review with meta-analysis found that the use of NPWT was associated with reduced LOS and NPWT may be more effective than standard therapy to manage SSIs.[44,45]

Caution is required when treating deep wounds that are packed. One continuous piece of rolled gauze is the preferred method for packing to avoid losing single dressings in the chest cavity. If foam is used for packing, write the number of pieces of foam used on the outermost dressing so that they can be removed with future dressing

changes. Further, if wounds require topical application of solutions, the large amount of open tissue will quickly absorb the fluids. Use of such products as povidone–iodine solution in these large wounds has resulted in iodine toxicity due to the large absorptive surface. Wounds that extend to or around the myocardium should be gently packed between heartbeats. Packing the wound tightly can constrict myocardial filling during diastole; therefore, the wound must be tucked loosely with gauze.

Chest Wall Reconstruction

Reconstruction of the chest wall can range from skin closure to chest wall stabilization. Defects following excision of a tumor, tissue loss from infection of the pleural space, and dehiscence of sternal wounds after coronary bypass grafting are examples of chest wall wounds that usually require reconstruction. Failure to stabilize the chest wall can lead to paradoxical chest motion with breathing, which results in compromised respiratory function. Prior to surgery, the patient's cardiac and pulmonary function, nutritional status, and wound bed should be maximized.

When a defect is limited to the skin and subcutaneous tissue, local skin flaps can frequently be used to close the wound. A skin flap can be rotated or advanced to cover the wound. The deltopectoral skin flap can also be used to cover chest wounds, although its blood supply is stretched to do so. The principal blood supply to the flap is from two large perforating arteries arising from the second or third intercostal space lateral to the sternal border. Therefore, any tension of the flap or chest can interfere with blood supply to the flap. The weight and torsion of the breast tissue or an obese chest can place tension on the flap, so a loose-fitting bra or binder should be used to support the tissue.

Loss of the sternum is especially complex because without the sternum, ribs are pulled inward with inspiration (e.g., flail chest). Patients can often tolerate the loss up to four ribs if the wound is closed with a muscle flap, such as the latissimus dorsi flap. Massive chest injuries or defects will require bony reconstruction; if bone is not used, acrylics or synthetic mesh can be used. However, the risks of

infection and rejection are ever present when synthetic materials are used.[46]

Practice Point

Immediately report to the surgeon any signs of flap ischemia, such as pale, dusky, mottled, or cool tissue.

When the chest wall defect is large, several muscles may be needed for reconstruction, depending on the location of the wound. The latissimus dorsi, pectoralis major, rectus abdominis, and trapezius are the most common muscles used for chest wall reconstruction.[46] The pectoralis muscle is commonly used because it is near the defect. Its blood supply is through the thoracoacromial artery, located in the axilla. The muscle is also supplied with perforating arteries from the internal mammary artery. If the pectoralis muscle is used, the patient will lose some ability to adduct and rotate the arm and may have some weakness with lifting. Due to the severity of the defect, this loss of donor site mobility is usually justified.[46]

Following surgery, vascular inflow and outflow must be closely monitored. Arterial insufficiency in the flap is a major hazard, and problems can occur from too much tension on the artery, hematoma in the underlying tissue, extravasated blood, increased blood viscosity, spasm of the artery, or injury or disease of the arterial vessels. Venous outflow problems occur from edema in the flap or compression of the veins. A flap with arterial inflow problems will appear pale and feel cool. Flaps with venous congestion will look engorged and purple and feel tense or full. Various monitoring options are available with none being shown clearly superior. They included Doppler exam, ViOptix (transcutaneous tissue oximetry), and clinical exam with turgor and capillary refill.

Pressure Injury Repair

Repairing a pressure injury for surgical closure is a process that involves multiple steps that begin well before surgery. After the pressure injury bed is clean, it can be closed surgically. However, before any decisions are made for surgery, such plans must be considered in light of the patient's situation, condition, and goals. While various surgical options may be technically possible, performing these operations for the right reasons is an essential first step. Surgery shouldn't be entered into lightly. For some patients, not closing the wound surgically may be the best decision. With proper nutrition, pressure redistribution, and local wound care, deep pressure injuries may remain stable for the duration of the patient's life. Adjunctive therapies, such as NPWT, electrical stimulation, and the use of bioengineered tissue products, may be considered to help with closure of these chronic wounds.[47]

Preparing the Patient for Surgery

Prior to any surgery, the patient's nutritional status and comorbidities must be controlled. Although operative blood loss is usually modest with this type of surgery, general anesthesia is commonly used and the patient must be able to tolerate the stress. General anesthesia is also used in paralyzed patients. The need for general anesthesia for repairs in these patients is often an area of question, with the patient and/or family asking "why do I need anesthesia if I can't feel below my waist?" This question should be answered in the following way: "Even though you may not feel pain, reflex arcs are frequently intact and the stress of the surgery and blood loss make blood pressure erratic." Many pressure injuries are repaired with the patient in a prone position, and general anesthesia is necessary to maintain a patent airway and oxygenation.

If malnutrition is the primary cause of nonhealing, surgery should be delayed until the patient has achieved positive nitrogen balance. Calorie counts provide clear data on actual intake of protein and calories, and adjustments can be made to reach ideal intake levels of 30 to 35 cal/kg of calories and 1.25 to 1.5 g/kg of protein.[48] Monitoring albumin is no longer considered a reliable marker due to albumin

being increasingly recognized as an acute-phase reactant and thereby lowered by any inflammatory processes. With a half-life of 3 days, serum prealbumin is another controversial marker for assessing visceral protein status but also affected by any inflammation. In patients with impaired renal function, prealbumin is artificially high because it is not removed with dialysis. Therefore, patients on dialysis should be classified as malnourished if the prealbumin level is less than 30 mg/dL, whereas the usual normal range is 16 to 35 mg/dL. More accurate nutritional information on protein status can benefit the healing potential for the patient. Indirect calorimetry is helpful to obtain more exact information on nutritional expenditure. It's important to recognize that patients who are malnourished are often deficient in vitamins (especially vitamin C) and minerals (especially zinc and iron); these supplements should be given throughout the course of care. The guidelines on pressure injury treatment[47] do not advise the routine use of vitamins and minerals for healing; they are recommended here only if the patient is deficient in them.

Practice Point

Because prealbumin is not removed during dialysis, use the lab value of less than 30 mg/dL to determine malnutrition.

Again, the need for aggressive nutritional support is a common area for clinical questions. Families and patients may view tube feeding as "artificial life support" and resist its use. The healthcare team needs to explain that unless the patient is eating adequately, there will be no reserve to heal. Acute wounds need more calories and protein to heal, and tube feeding (or hyperalimentation) should be considered as a short-term method to increase the likelihood of healing (see Chapter 10, Nutrition and Wound Care, for additional information).

If the wound is due primarily to pressure (such as an ischial pressure injury in a paraplegic patient), pressure must be redistributed

both before and after surgery. Long-term plans for continued pressure redistribution must be included in the operative plan, such as fitting the patient for a wheelchair and appropriate off-loading device and teaching the patient pressure-relief techniques to avoid high rates of recidivism.[49]

If the wound is due to a combination of erosion, shear, and pressure (such as skin breakdown on the sacrum in a patient with dyspnea and incontinence), all contributing factors need to be addressed. Similarly, long-term pressure reduction and proper skin care management must be included in the care plan. There are a finite number of available flaps; therefore, all efforts must be directed to prevent recurrent injuries, especially through education of the patient. The social network of the patient with a spinal cord injury has been shown to be very crucial in maintaining a healed wound.[49]

Wound infection may not be evident from the surface appearance of the wound. The presence of osteomyelitis should be considered if the bone feels rough or soft and ruled out in all stage 4 pressure injuries. MRI is usually helpful to identify the infection. Biopsy after adequate debridement remains the gold standard in diagnosing osteomyelitis if still suspected and the above studies are negative.

Other infections must also be controlled prior to surgery. Urinary tract infections are common in people with diabetes, elderly women, patients with catheters, and those with sacral wounds. Urosepsis is a serious complication and should be considered as the cause of malnutrition and changes in mental status or vital signs malnutrition. Chronic urinary antibiotics may be needed for persistent urinary tract infections. Wound infection can also lead to sepsis[50] (see Chapter 7, Wound Bioburden and Infection).

Spasms are often a contributing factor to both shear and friction injuries and may also complicate a postoperative course by putting undo tension on wound closure sites. Spasms are common after spinal cord injury due to loss of supraspinal inhibitory pathways. The higher the level of spinal cord injury, the more likely spasms will occur. People with cervical injury have almost a 100% chance of spasm compared with those with injury in the lower thoracic or lumbar spine, who have a 50% chance of spasm. Spasm must be controlled prior to

surgery with medications such as baclofen (Lioresal) or dantrolene (Dantrium). Botulinum toxin (Botox) can also be injected into the muscle to attempt to reduce spasm; however, the efficacy of this treatment is unrecorded. Spastic limbs may lead to wound dehiscence and postoperative wound complications and therefore must also be controlled.

Contracture develops in patients with long-standing denervation caused by tightening of the muscle and joint capsules. Because hip flexors are so strong, hip contraction is common and can make positioning difficult, with bony prominences resting on each other and leading to ulcers. Contractures can be minimized with proper, persistent positioning, splinting, and a program of aggressive (often passive) range-of-motion exercises. Patients with significant contracture can't be placed in a supine position; if surgery is performed on one hip, only one side will be available as a turning surface, which will require a sophisticated pressure redistribution system to prevent complications or undue pressure at the other hip. A defined turning schedule is essential in these cases. If contractures can be released via tenotomy, the wound may heal. However, leaving a limb flaccid after tenotomy may leave the patient bedridden if he or she relies on the spastic limb for postural support while moving.

Preparing the Wound for Surgery

Debridement of nonviable tissue is an important first step in the treatment of full-thickness necrotic pressure injuries or infected, dehisced surgical wounds. Breast debridement of adherent eschar in pressure injuries is advised to reduce the wound bioburden and risk of sepsis (Fig. 20-4). The true stage of a pressure injury can't be determined until adequate debridement has been performed because the true depth of the wound is obscured. Debridement can result in extensive wounds. For example, pelvic pressure injuries can extend to the scapula and to the vagina or trochanters. Removal of necrotic tissue may enhance the wound healing cascade and diminishes the risk of infection (see Chapter 8, Wound Debridement). Dry and stable gangrene should usually not be removed from foot ulcers in patients with impaired arterial supply. Changing a closed and stable wound to an open and ischemic wound creates a greater problem.

Figure 20-4. Wound dehiscence following breast reduction. (Photo courtesy of Barbara Delmore.)

Wound debridement is most thoroughly completed in the operating room. Bedside debridement may be used to unroof hard eschar but can seldom be completed to the level of a clean wound bed. Enzymatic debridement will often provide reasonable wound debridement but may take many weeks. However, in the patient who is a poor surgical risk, enzymatic agents are a good alternative.

Determining which wounds would benefit from debridement is the first step. Necrotic tissue is generally understood to be a healing deterrent because it allows infection. Necrotic tissue has no blood supply, so antibiotics and antibodies are not present in it. The wound and surrounding tissue should be examined fully to assess for fluid collection, abscess formation, and extensions into surrounding tissue.

Whether or not heels should be debrided remains controversial. Most clinicians feel that stable, dry, adherent eschar on ischemic heels should not be debrided, and recent data show healing of heel ulcers when eschar is left intact.[51] The foot, especially the posterior heel, has very limited blood flow and only a small amount of subcutaneous fat. Once the underlying fatty tissue is exposed to the

environment, it may become infected quickly due to desiccation and its limited blood flow. If the bone is exposed during debridement, osteomyelitis may be an inevitable occurrence. Stable, dry eschar that has no openings should be left intact, assessed often, and off-loaded completely by placing pillows under the calves or floating the heel in orthotic splints/boots. The phrase “float the heels” is an appropriate order to convey the idea that no pressure is to be applied to the heels. If the eschar softens or breaks or the tissue around the ulcer becomes fluctuant or inflamed, the tissue should be excised to prevent deeper bacterial invasion and sepsis. Chemical debridement may be needed to continue the process of wound bed preparation. Moist wound healing techniques should be instituted at this time. Off-loading should continue throughout the course of treatment.

Flaps for Pressure Ulcer Repair

Large areas of the skin or the skin and muscle may be missing from a pressure injury. Surgical repair options depend on what kind of tissue needs replacement. If the skin is missing on a sacral wound, for example, rotational flaps of skin or skin and fascia can be rotated to close the wound. If the muscle is missing, such as in a deep stage 4 pressure injury, the gluteus may be used to provide padding and protection of the bony structures. The muscle moved into the wound doesn’t function as muscle; it atrophies over time due to denervation. Muscle tissue provides padding and robust blood supply to combat osteomyelitis. Muscle also carries more skin with its named blood vessels, so more skin can be moved without ischemia developing (Fig. 20-5, Table 20-1).

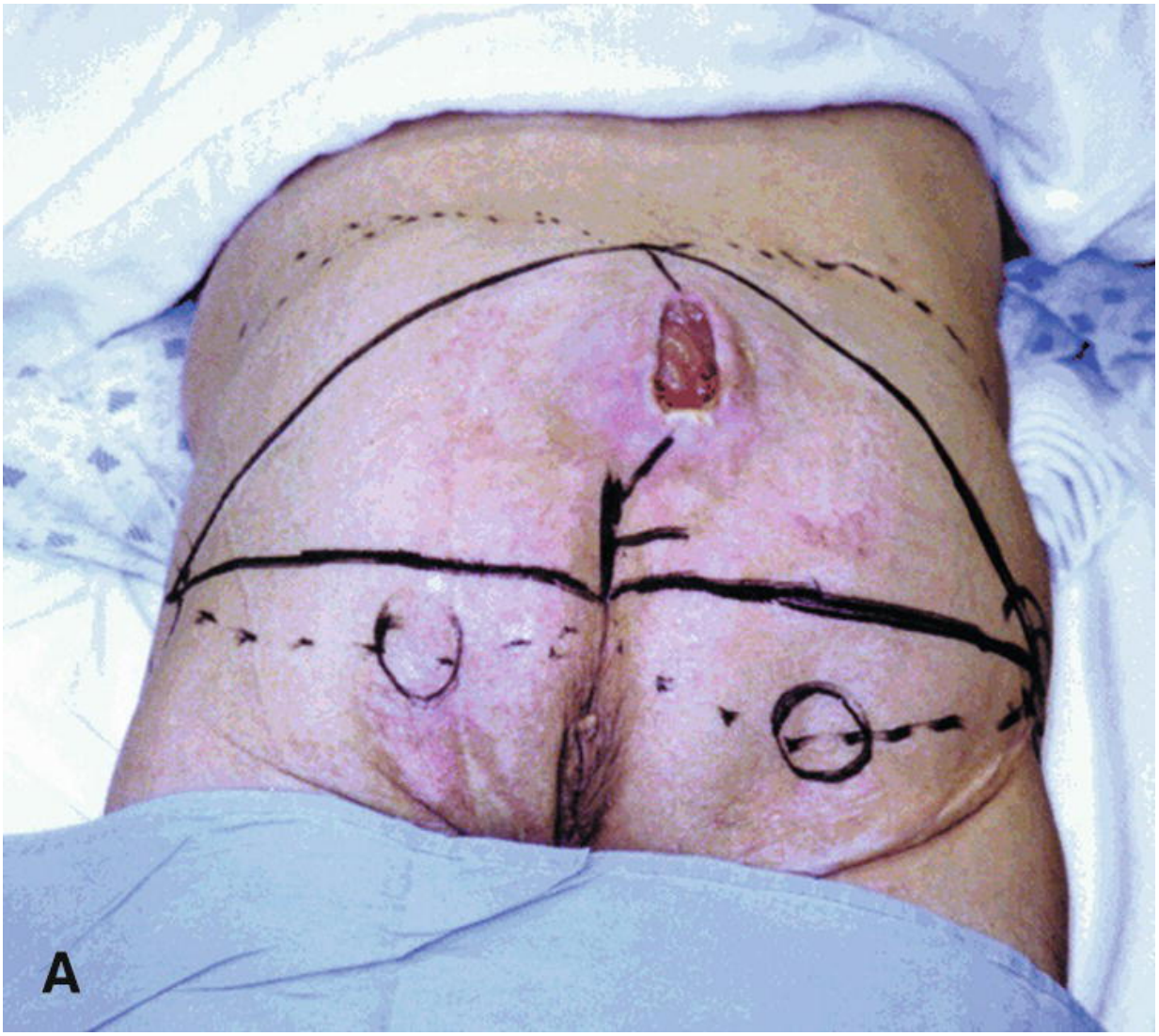
A

Figure 20-5. Surgical closure of a pressure ulcer. **(A)** This photograph shows markings made for gluteal fasciocutaneous flaps for surgical closure of a stage 2 pressure injury. **(B)** Shown here is surgical closure of the same pressure injury (Photo courtesy of Jonathan S. Black.).

Table 20-1 Flaps for Pressure Ulcer Repair

Pressure Ulcer Site	Muscle Flap Options	Skin or Fasciocutaneous Flap Options
Sacral ulcer	• Gluteus maximus (superiorly or inferiorly based) or V–Y advancement	• Tensor fascia lata (may retain sensation)
Ischial ulcer	• Gluteus maximus (superiorly based) • Biceps femoris • Semimembranosus • Semitendinosus • Gracilis	• Transverse or vertical lumbosacral • Posterior thigh advancement flap with skin graft of donor site • Posterior V–Y advancement flap • Medial thigh rotation flap
Trochanteric ulcer	• Tensor fascia lata (may retain sensation)	

This chart presents common closure options for pressure ulcers at common sites.

Postoperative Care

The transplanted muscle flap requires adequate perfusion of arterial blood and drainage of venous blood to survive. Because a limited number of flaps are available to reconstruct any wound, the consequence of flap failure is great. Early pressure ulcer flap failure is most commonly due to pressure or osteomyelitis. Flaps that have impaired arterial flow appear pale, have poor to absent capillary refill, and show sluggish bleeding when lanced. These wounds require quick restoration of their arterial supply by opening the wound and examining the arterial inflow. Occasionally, arterial spasm is the culprit. Spasm can be treated by positioning the flap dependently and warming the area.

Practice Point

Early signs of arterial flap failure are:

- pale color
- poor or absent capillary refill
- sluggish bleeding when lanced
- loss of audible pulses by Doppler.

Flaps that have impaired venous drainage appear dark blue and swollen. The problem is seldom a faulty anastomosis site; usually, it results from the sluggish exit of venous blood. Venous congestion can be treated by elevation and application of leeches to drain excess blood from the flap. NPWT may also be recommended to decrease fluid collection. Drains are commonly used to empty fluid accumulation in dead space and can be left in place for a week or longer until drainage has subsided.

Tension on the incision line can lead to dehiscence. Bolster dressings are typically used to close the wound with little tension. Suture lines are slow to heal, especially in the denervated patient, and sutures are left in place for at least 3 weeks. Due to poor approximation and tensile strength, great caution must be used when moving the patient to avoid pulling on the suture line. It's possible to tear open a late-stage surgical repair, which commonly leads to complete flap failure. Small areas of dehiscence are often left to heal via granulation tissue.

Pressure relief is crucial following flap repair. Surgeons usually prescribe bed rest on an air-fluidized beds or low-air-loss beds for 2 to 6 weeks. Be sure the patient is not removed from the bed without the surgeon's permission (e.g., sent for an x-ray procedure). Large skin flaps are especially prone to failure because of tension on the distal edges of the flap. If fecal incontinence is likely, the patient may be placed on a low-residue diet and constipating medications. Diverting colostomy may be required prior to flap closure in extreme cases to prevent contamination of the surgical site with stool. Usually, bedpans are contraindicated for the first few weeks.

The patient must be compliant with off-loading strategies after a surgical flap for wound closure to prevent breakdown of the surgical repair. This involves not only immediate postoperative pressure redistribution but continued interventions of pressure relief and reduction after complete healing has occurred, especially in patients confined to wheelchairs. Proper chair cushions and weight shifts are essential.[49]

When describing healing of flaps, the wound is a surgical wound, not a pressure injury. Its healing is primary and not secondary with

granulation tissue.

Leg Reconstruction

Tissue defects of the leg can be reconstructed with muscle flap covered with skin grafts, myocutaneous flaps, or free flaps, depending on the location of the wound and available donor tissue. Attempts are made to salvage the leg unless there is irreversible nerve or vessel damage. Major soft tissue injuries with or without bone involvement provide an environment favorable for infection. Wound care is commonly completed in the operating room, where debridement can be performed in a sterile, controlled atmosphere. Definitive wound coverage often includes the rectus abdominis, gluteus, rectus femoris, gastrocnemius, and soleus muscle.

Following surgery, it's imperative to monitor the flap for signs of vascular compromise. These situations are emergent; without immediate intervention, the limb may be lost due to ischemia. Unusual findings must be reported promptly and accurately to the attending surgeon.

Practice Point

Monitor for the following signs of vascular compromise:

- Pallor and coolness
- Lack of pulses
- Pain with movement
- Slow or absent capillary refill
- Inability to move extremity
- Abnormal sensations (pins and needles)

Of note, synthetic dermal templates (e.g., Integra) have emerged as a nonflap option to cover small areas of exposed bone or tendon. They have particularly shown use in lower extremity wounds with limited local flap options. Combination with NPWT has

shown optimal rates of revascularizing the template, which then provides an adequate bed for skin grafting and a simplified closure.[52]

Necrotizing Fasciitis

Necrotizing fasciitis, also known as synergistic gangrene and “flesh-eating bacteria” infection, is a rapidly progressive soft tissue infection. Although beta-hemolytic *Streptococcus pyogenes* is the most common causative organism, no single organism is responsible for the infection. Frequently, necrotizing fasciitis is caused by two organisms acting in concert, called synergistic gangrene. Gram-positive bacteria (including methicillin-resistant *Staphylococcus aureus*), gram-negative bacteria, anaerobic bacteria (including gas gangrene caused by *Clostridium perfringens*), marine *Vibrio*, and fungi have been identified. Necrotizing fasciitis caused by beta-hemolytic streptococcal organisms is highly sensitive to antibiotics. However, antibiotics don’t penetrate necrotic tissue, so delays in treatment can lead to a 73% mortality.[53]

Necrotizing fasciitis appears to develop following a breach in the integrity of a mucous membrane barrier, especially in the abdomen, perineum, and extremities. Sacral pressure injuries can develop necrotizing soft tissue infections also. A malignancy may also give rise to a portal of entry. In males, leakage into the perineal region can result in a syndrome called Fournier’s gangrene, which is characterized by massive swelling and tissue loss of the scrotum and penis with extension into the perineum, the abdominal wall, and the legs.

Pain out of proportion to the size and extent of the skin wound is the hallmark sign. Early presentation also includes a reddened, painful, swollen area of cellulitis and fever. More generalized swelling develops and is followed by brawny edema. With progression, dark red induration of the epidermis appears, along with bullae (filled with blue or purple fluid). Later, the skin becomes friable and takes on a bluish, maroon, or black color. By this stage, thrombosis of blood vessels in the dermal papillae is extensive. Extension of infection to

the level of the deep fascia causes this tissue to take on the brownish–gray appearance of frank gangrene. The skin is normally a very effective barrier preventing bacteria from invading the body; likewise, immune cells in the skin are also able to trap organisms, preventing rapid spread along fascial planes and through venous channels and lymphatics. Patients in the later stages are toxic and frequently manifest septic shock and multiorgan system failure. Progression may be rapid. Skin inflammation is rapidly followed by necrosis of superficial fascia, subcutaneous fat, and, in some cases, muscle. Necrotizing fasciitis is commonly seen in conjunction with severe systemic toxicities.

Practice Point

The hallmark of necrotizing fasciitis is pain out of proportion to physical findings.

Diagnosis and Treatment

Aspiration cultures from the wound edge or punch biopsy with frozen section may be helpful if the results are positive, but false-negative results occur in approximately 80% of cases. There is some evidence that aspiration alone may be superior to injection and aspiration using normal saline solution. Extensive gas can be seen along the fascial planes on radiograph. However, deep biopsy and frozen section histopathology often confirm the diagnosis. Frozen sections are especially useful in distinguishing necrotizing fasciitis from other skin infections such as toxic epidermal necrolysis.

Practice Point

In cases of suspected necrotizing fasciitis, myositis, or gangrene, early and aggressive surgical exploration is necessary to:

- visualize deep structures
- remove necrotic tissue
- open compartments to decrease pressure
- obtain tissue for Gram stain for aerobic and anaerobic organisms.

Repeated debridements are usually necessary until all devitalized tissue is removed, and early debridement has been associated with improved outcomes.[54] Intravenous broad-spectrum antibiotics are started, pending more specific culture results. Due to the complexity of these wounds and patients, consultation with infectious disease specialists is imperative. The current empirical antimicrobial regimens advocate use of piperacillin–tazobactam with clindamycin and ciprofloxacin for infections from a combination of aerobes and anaerobes. For infections from group A streptococcus or *Staphylococcus aureus*, penicillin with clindamycin is recommended. For clostridial infections, combination of penicillin with clindamycin is effective. The final course of antimicrobial therapy should be based on the culture sensitivity reports. In cases of fungal infections, intravenous amphotericin B is given. Appropriate empirical antibiotic treatment for necrotizing fasciitis from group A streptococcal organisms is commonly clindamycin plus penicillin G and cephalosporin (first or second generation). Mixed aerobic–anaerobic infections can be treated with ampicillin and sulbactam, cefoxitin, or combinations of either clindamycin, metronidazole, and ampicillin or ampicillin, sulbactam, and carbapenem.[55] Hyperbaric oxygen treatment may also be useful as many of these infections have an anaerobic or microaerophilic component. Antibiotic treatment should be continued until all signs of systemic toxicity have resolved, all

devitalized tissue has been removed, and granulation tissue has developed. Multiorgan system failure is not uncommon.

Following debridement, wounds are left open and packed. The packing can be extensive and may be completed in the operating room with daily debridement. Definitive treatment may include amputation or extensive skin grafting after the infection is under control and the wounds show evidence of granulation. It's essential that the dressings stay moist to prevent desiccation of tissue. It may be helpful to use products with a higher absorptive capacity than gauze to manage fluid from draining wounds.

In one retrospective study, patients who survived an episode of necrotizing fasciitis had an increased risk of premature death due to infection, such as pneumonia, cholecystitis, urosepsis, or sepsis.[56] Women died much earlier than men in the study. In our experience, patients with necrotizing fasciitis have an increased frequency of colon cancer, and colonoscopy prior to dismissal has become part of the routine care provided.

Summary

Understanding the etiology of complex wounds is a critical first step in developing an effective treatment plan. Recognition of the unique characteristics of each of these wound categories will enable the clinician to correctly identify the wound. The goal of care for acute wounds is wound closure with the return of form and function. Use of the reconstructive ladder can assist clinicians in the decision-making process. Surgical management may be part of the care plan for complex acute and chronic wounds. Monitoring for signs of failure is imperative for patients with skin flaps and grafts.

PATIENT SCENARIO

Clinical Data

Andre is a 22-year-old man who became a T12 paraplegic following a motor vehicle crash 2 years ago. Andre had been the driver of the car, and his best friend was killed in the crash. Andre has been struggling with his residual paralysis and accepting responsibility for his injuries and his friend's death. He was treated initially at a trauma center for repair of a fractured spine, followed by rehabilitation at a spinal cord center. He often expressed the hope to "walk again" doing "whatever it takes." He uses a wheelchair with a gel cushion to allow for sliding transfers.

This admission to acute care began with treatment in the emergency department for fever and chills. He was diagnosed with sepsis, the source of which was determined to be his ischial pressure ulcers. At the time of admission, the wounds were found to be dressed with newspaper. Andre said he did not have the money to buy dressings and uses paper towels or newspaper to absorb the drainage. He has no other chronic illnesses. He is protein malnourished and smokes 1 to 1½ packs of cigarettes daily.

Examination of Andre's wounds reveals extensive pressure injuries throughout the perineum (Fig. 20-6). Both unstageable ischial

pressure injuries and healing pressure ulcers are present. Surgical consultation is recommended.

Figure 20-6. Extensive pressure ulcers throughout the perineum in a 22-year-old paraplegic man.

Case Discussion

Let's consider the following four questions as the basis for our clinical decision-making regarding Andre's plan of care:

1. **What surgical treatment is likely to be recommended now?** Because the source of his sepsis is his wounds, these pressure injuries need to be debrided. Ideally, the debridement should be done in the operating room; however, if he is hypotensive from the sepsis, the wounds could be unroofed at the bedside.

Eventually, the wounds will need to be evaluated to determine whether osteomyelitis is present. Infectious disease consultation should be obtained. Once certain anaerobic infection is controlled, NPWT could be used to promote healing.

2. **What type of pressure redistribution system is needed in the bed and in the chair?** An integrated bed system with low air loss and patient movement or an air-fluidized system is needed. While these pressure injuries could be seen as affecting only one turning surface, the use of a standard foam mattress will likely not be adequate because his risk for new ulcers is also high enough to justify the expenditure for an upgraded surface.
3. **Is flap closure advisable? Why or why not?** Technically, there are flaps that could be raised to close these injuries. However, it would likely be a multistage procedure and the flaps can only be used once. Unless Andre becomes more compliant with an off-loading program, the flaps are likely to fail. He has obviously had several pressure injuries and even a surgical debridement in the past 2 years. He may still be angry over the physical impact on his body and would benefit from mental health evaluation. A quick surgical fix to close these wounds would likely not benefit him in the long run.
4. **What social support is imperative in order for these wounds to stabilize? Heal?** Andre's social support network needs to be explored. If he lives alone, he might benefit from a group home with other persons confined to wheelchairs. The positive role modeling from others with spinal cord injury may be able to engage him to do off-loading and examine his skin for signs of breakdown. Social services should be working with Andre to ensure that he has a medical benefit program that can provide appropriate supplies. He also needs to be evaluated by physical and occupational therapists to assess his wheelchair, wheelchair cushion, methods of transfer, and off-loading, and other potential rehabilitation strategies. Impact of smoking on wound healing needs to be explored with Andre. Even if he is

unwilling to stop smoking, he might be agreeable to reducing the amount of smoking. This could be a good place to start.

Show What You Know

1. **A wound that has been open and then closed via a surgical procedure is considered to have healed via:**
    A. surgical closure.
    B. tertiary intention.
    C. primary intention.
    D. secondary intention.

2. **A newly created suture line should remain covered for at least 48 hours so that:**
    A. the amount of drainage can be detected.
    B. the patient does not touch the suture line.
    C. the natural skin barrier has time to form.
    D. the patient does not experience pain.

3. **There are how many types of SSIs?**
    A. Five—superficial incisional, deep incisional, deep incision primary, deep incision secondary, and organ/space
    B. Two—superficial incisional and deep incisional
    C. Four—superficial incisional, deep incisional, deep incision primary, and deep incision secondary
    D. Three—superficial incisional, deep incisional, and organ/space

4. **Which of the following situations depicts the use of the simplest method for closure of a wound?**
    A. A skin graft of a venous stasis ulcer
    B. Secondary healing of a calcium extravasation
    C. Primary closure for a sternal incisional dehiscence
    D. A free fasciocutaneous flap for a stage 3 pressure injury

5. **Which of the following wounds might require a musculocutaneous flap for closure?**
    A. Large burn on the face
    B. Calciphylaxis of the lower legs

C. Radiation necrosis of the chest wall
D. A stage 2 pressure injury on the trochanter

6. **Which of the following signs might indicate arterial impairment in a flap?**
   A. Pain and coolness
   B. Pallor and warmth
   C. Slow capillary refill and pain
   D. Slow capillary refill and pallor

7. **You discharged a patient with ischial pressure injuries from the hospital after 6 weeks of local wound care. Upon return to the clinic, the wounds have recurred and are necrotic. Which of these causes of recurrence should be investigated first?**
   A. Presence of urinary incontinence
   B. Inadequate pressure redistribution in seating
   C. Deterioration of nutritional status
   D. Development of ischial osteomyelitis

Answers to these questions are found on pages 686 and 687.

References

1. Delmore, B., Cohen, J.M., O'Neill, D., et al. Reducing Postsurgical Wound Complications: A Critical Review. Advances in Skin & Wound Care 30(6):272-85, 2017. doi: 10.1094/PDIS-92-9-1364A.
2. Young, A., McNaught, C.E. The Physiology of Wound Healing. Surgery 29(10):475-9, 2011. doi: 10.1016/j.mpsur.2011.06.011.
3. Gantwerker, E.A., Hom, D.B. Skin: Histology and Physiology of Wound Healing. Clinics in Plastic Surgery 39(1):85-97, 2012. doi: 10.1016/j.cps.2011.09.005.
4. Holloway, S., Harding, K., Stechmiller, J., et al. Acute and Chronic Wound Healing. In S. Baranoski, E.A. Ayello, eds. Wound Care Essentials: Practice Principles, 4th ed. Philadelphia, PA: Wolters Kluwer, 2016:82-98.
5. Geronemus, R.G., Robins, P. The Effect of Two New Dressings on Epidermal Wound Healing. Journal of Dermatologic Surgery and Oncology 8(10):850-2, 1982. doi: 10.1111/j.1524-4725.1982.tb03992.x.
6. Orgil, D., Blacno, C. Introduction: Development of Skin Substitutes. In D.P. Orgill, C. Blanco, eds. Biomaterials for Treating Skin Loss. Florida: Woodhead Publishing Limited, 2009:3-8.
7. Mehendale, F., Martin, P. The Cellular and Molecular Events of Wound Healing. In V. Falanga, ed. Cutaneous Wound Healing, 1st ed. London: Martin Duntz, 2001:15-37.
8. Saxena, V. Biomechanics of Skin. In D. Orgill, C. Blanco, eds. Biomaterials for Treating Skin Loss. Florida: Woodhead Publishing Limited, 2009:18-24.
9. Ban, K.A., Minei, J.P., Laronga, C., et al. American College of Surgeons and Surgical Infection Society: Surgical Site Infection Guidelines, 2016 Update. Journal of American College of Surgeons 224(1):59-74, 2017. doi: 10.1016/j.jamcollsurg.2016.10.029.
10. Mangram, A.J., Horan, T.C., Pearson, M.L., et al. Guideline for Prevention of Surgical Site Infection, 1999. Centers for Disease Control and Prevention (CDC) Hospital Infection Control Practices Advisory Committee. American Journal of Infection Control 27(2):97-132, 1999; quiz 133-4; discussion 96. doi: 10.1016/S0196-6553(99)70088-X.
11. Stroncek, J.D., Bell, N., Reichert, W.M. Instructional PowerPoint Presentations for Cutaneous Wound Healing and Tissue Response to Sutures. Journal of Biomedical Materials Research. Part A 90(4):1230-8, 2009. doi: 10.1002/jbm.a.32158.
12. Toon, C.D., Lusuku, C., Ramamoorthy, R., et al. Early Versus Delayed Dressing Removal After Primary Closure of Clean and Clean-Contaminated Surgical Wounds. Cochrane Database of Systematic Reviews 9(9):CD010259, 2015. doi: 10.1002/14651858.CD010259.pub3.
13. Stroncek, J.D., Reichert, W.M. Overview of Wound Healing in Different Tissue Types. In W.M. Reichert, ed. Indwelling Neural Implant Strategy Contend with

Vivo Environment. Boca Raton, FL: CRC Press/Taylor & Francis, 2008.
14. Szpaderska, A.M., Zuckerman, J.D., DiPietro, L.A. Differential Injury Responses in Oral Mucosal and Cutaneous Wounds. Journal of Dental Research 82(8): 621-6, 2003. doi: 10.1177/154405910308200810.
15. Winter, G. Epidermal Regeneration Studied in the Domestic Pig. Fundamentals of Wound Management. 1979.
16. Rippon, M., Davies, P., White, R. Taking the Trauma Out of Wound Care: The Importance of Undisturbed Healing. Journal of Wound Care 21(8):359-60, 362, 364-8, 2012. doi: 10.12968/jowc.2012.21.8.359.
17. Sibbald, R.G., Goodman, L., Woo, K.Y., et al. Special Considerations in Wound Bed Preparation 2011: An Update. Advances in Skin & Wound Care 24(9):415-36, 2011. doi: 10.1097/01.ASW.0000405216.27050.97.
18. Jones, V.J. The Use of Gauze: Will It Ever Change? International Wound Journal 3(2):79-86, 2006. doi: 10.1111/j.1742-4801.2006.00215.x.
19. Cutting, K.F. Impact of Adhesive Surgical Tape and Wound Dressings on the Skin, with Reference to Skin Stripping. Journal of Wound Care 17(4):157-8, 160-2, 2008. doi: 10.12968/jowc.2008.17.4.28836.
20. Centers for Medicare & Medicaid Services. Hospital-Acquired Conditions. Published 2008. Available at: https://www.cms.gov/Medicare/Medicare-Fee-for-Service-Payment/HospitalAcqCond/Hospital-Acquired_Conditions.html. Accessed March 27, 2019.
21. Anderson, D.J., Podgorny, K., Berríos-Torres, S.I., et al. Strategies to Prevent Surgical Site Infections in Acute Care Hospitals: 2014 Update. Infection Control and Hospital Epidemiology 35(6):605-27, 2014. doi: 10.1086/676022.
22. Umscheid, C.A., Mitchell, M.D., Doshi, J.A., et al. Estimating the Proportion of Healthcare-Associated Infections That Are Reasonably Preventable and the Related Mortality and Costs. Infection Control and Hospital Epidemiology 32(2):101-14, 2011.
23. Bratzler, D.W., Dellinger, E.P., Olsen, K.M., et al. Clinical Practice Guidelines for Antimicrobial Prophylaxis in Surgery. American Journal of Health-System Pharmacy 70(3):195-283, 2013. doi: 10.2146/ajhp120568.
24. Magill, S.S., Edwards, J.R., Bamberg, W., et al. Multistate Point-Prevalence Survey of Health Care-Associated Infections. New England Journal of Medicine 370(13):1198-208, 2014. doi: 10.1056/NEJMoa1306801.Multistate.
25. Merkow, R.P., Ju, M.H., Chung, J.W., et al. Underlying Reasons Associated with Hospital Readmission Following Surgery in the United States. Journal of the American Medical Association 313(5):483-95, 2015. doi: 10.1001/jama.2014.18614.
26. Berenguer, C.M., Ochsner, M.G., Lord, S.A., et al. Improving Surgical Site Infections: Using National Surgical Quality Improvement Program Data to Institute Surgical Care Improvement Project Protocols in Improving Surgical Outcomes. Journal of American College of Surgeons 210(5):737-41, 2010. doi: 10.1016/j.jamcollsurg.2010.01.029.

27. Berríos-Torres, S.I., Umscheid, C.A., Bratzler, D.W., et al. Centers for Disease Control and Prevention Guideline for the Prevention of Surgical Site Infection, 2017. JAMA Surgery 152(8):784-91, 2017. doi: 10.1001/jamasurg.2017.0904.
28. Tillman, M., Wehbe-Janek, H., Hodges, B., et al. Surgical Care Improvement Project and Surgical Site Infections: Can Integration in the Surgical Safety Checklist Improve Quality Performance and Clinical Outcomes? Journal of Surgical Research 184(1):150-6, 2013. doi: 10.1016/j.jss.2013.03.048.
29. Meeks, D.W., Lally, K.P., Carrick, M.M., et al. Compliance with Guidelines to Prevent Surgical Site Infections: As Simple As 1-2-3? *American Journal of Surgery* 201(1): 76-83, 2011. doi: 10.1016/j.amjsurg.2009.07.050.
30. Allegranzi, B., Zayed, B., Bischoff, P., et al. New WHO Recommendations on Intraoperative and Postoperative Measures for Surgical Site Infection Prevention: An Evidence-Based Global Perspective. Lancet Infectious Diseases 16(12):e288-303, 2016. doi: 10.1016/S1473-3099(16)30402-9.
31. Leaper, D., Burman-Roy, S., Palanca, A., et al. Prevention and Treatment of Surgical Site Infection: Summary of NICE Guidance. BMJ 337(7677):a1924, 2008. doi: 10.1136/bmj.a1924.
32. Levine, J. “Historical Notes on Pressure Ulcers: The Cure of Ambrose Pare.” Decubitus 5(2):23-4, 1992.
33. Edlich, R.F., et al. “Revolutionary Advances in the Management of Traumatic Wounds in the Emergency Department During the Last 40 Years: Part I.” Journal of Emergency Medicine 17(12):1284-302, 1988.
34. Scheppach, W. “Abdominal Compartment Syndrome.” Best Practice & Research Clinical Gastroenterology 23(1):25-33, 2009.
35. Paylidis, T.E., et al. “Complete Dehiscence of the Abdominal Wound and Incriminating Factors.” European Journal of Surgery 167(5):351-5, 2001.
36. Jang, J.Y., et al. “Application of Negative Pressure Wound Therapy in Patients with Wound Dehiscence After Abdominal Open Surgery: A Single Center Experience.” Journal of Korean Surgical Society 85(4):180-4, October 2013.
37. Argenta, L., et al. “Vacuum-assisted Closure: State of the Clinical Art.” Plastic and Reconstructive Surgery 117(7S):127S-42S, 2006.
38. van Ramshorst, G.H., et al. “Abdominal Wound Dehiscence in Adults: Development and Validation of a Risk Model.” World Journal of Surgery 34(1):20-7, January 2010.
39. Doughty, D. “Preventing and Managing Surgical Wound Dehiscence.” Home Healthcare Nurse 22:364-67, 2005.
40. Bell, P.L., Gabriel, V. “Evidence Based Review for the Treatment of Post-Burn Pruritus.” Journal of Burn Care & Research 30:55-61, 2009.
41. Breuing, K.H., et al. “Early Experience Using Low-frequency Ultrasound in Chronic Wounds.” Annals of Plastic Surgery 55(2):183-87, 2002.
42. Fowler, V., et al. “Clinical Predictors of Major Infections after Cardiac Surgery.” Circulation 112(9 Suppl): 1358-65, 2005.

43. Ahumada, L.A., et al. “Comorbidity Trends in Patients Requiring Sternotomy and Reconstruction.” Annals of Plastic Surgery 54(3):264-8, 2005.
44. Petzina, R. “Negative Pressure Wound Therapy for Post-Sternotomy Mediastinitis Reduces Mortality Rate and Sternal Re-Infection Rate Compared to Conventional Treatment.” European Journal of Cardiothoracic Surgery 38(1):110-3, July 2010.
45. Pan, A., et al. “Negative Pressure to Treat Surgical Site Infections, with a Focus on Post-Sternotomy Infections: A Systematic Review and Meta-Analysis.” Infection 41(6):1129-35, December 2013.
46. Song, D., Roughton, M. “Reconstruction of the Chest.” In D. Song, ed. Plastic Surgery, 3rd ed. St Louis, MO: Elsevier, 2013:249-55.
47. Black, S., Black, J., Brem, H. Surgery for Pressure Ulcers. In EPUAP-NPUAP Guidelines for Prevention and Treatment of Pressure Ulcers. Washington, DC: National Pressure Ulcer Advisory Panel, 2009.
48. National Pressure Ulcer Advisory Panel, European Pressure Ulcer Advisory Panel and Pan Pacific Pressure Injury Alliance; Haesler, E., eds. Prevention and Treatment of Pressure Ulcers: Clinical Practice Guideline. Osborne Park, Western Australia: Cambridge Media, 2014.
49. Krause, J.S., Broderick, L. “Patterns of Recurrent Pressure Ulcers after Spinal Cord Injury: Identification of Risk and Protective Factors 5 or More Years after Onset.” Archives of Physical Medicine and Rehabilitation 85:1257-64, 2004.
50. Mizokami, F., et al. “Necrotizing Soft Tissue Infections Developing from Pressure Ulcers.” Journal of Tissue Viability 23(1):1-6, February 2014.
51. Molnar, J., DeFranzo A., Hadaegh A., et al. Acceleration of Integra Incorporation in Complex Tissue Defects with Subatmospheric Pressure. Plastic and Reconstructive Surgery 113(5):1339-46, April 15, 2004.
52. Shannon, M. “A Retrospective Descriptive Study of Nursing Home Residents with Heel Eschar or Blisters.” Ostomy Wound Management 59(1):20-7, 2013.
53. Trent, N., Kirsher, R.S. “Necrotizing Fasciitis.” Wounds 14(8):284-92, 2002.
54. Sugihara, T., et al. “Impact of Surgical Intervention Timing on the Case Fatality Rate for Fournier’s Gangrene: An Analysis of 379 Cases.” British Journal of Urology 110:E1096-100, December 2012.
55. Endorf, F., et al. “Necrotizing Soft-Tissue Infections: Clinical Guidelines.” Journal of Burn Care and Research 30(5):769-75, September-October 2009.
56. Mishra, S.P., et al. “Necrotizing Soft Tissue Infections: Surgeon’s Perspective International.” Journal of Inflammation 2013:1-7, 2013.

21

Tube, Drain, and Fistula Management

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We gratefully acknowledge the contributions of Paula Toth, MSN, RN, CWOCN, CNS, for her work on previous editions of this chapter.

Objectives

After completing this chapter, you'll be able to:

- describe the purpose of tubes and drains
- state management considerations of enterocutaneous fistulas
- discuss primary management concerns for drains, tubes, and fistulas.

Introduction

The care and management of percutaneous tubes and of various fistula types creates unique challenges for the clinician including fluid and electrolyte balance, skin integrity concerns, potential for infection, and nutritional concerns.[1] The wound clinician is often consulted to provide management options addressing resulting skin integrity complications and, in some cases, the direct wound care associated with a tube or fistula. It is not uncommon for the person with a chronic wound to have complications preventing wound closure and require additional assessment to identify healing barriers, management options, and direct wound care. The care and management of any chronic wound requires a holistic approach that considers how chronic disease, causative factors, psychosocial support, and the skill of the care provider impact care and the ability to heal.

Tubes and Drains

A percutaneous tube is placed into a body organ or space through a cutaneous entry point.[1,2] Depending on the disease process, condition, or purpose of the tube, placement will promote drainage, decompression, lavage, medication administration, fluid instillation, or the means to administer nutritional support.[1–3] Additionally, symptom management, palliative care, and pain management can be addressed through access to different anatomical locations.[2] The care and management of a specific percutaneous tube begins with the anatomical location of the tube, manufacturer's guidelines for care, anatomical anomalies that can interfere with stabilizing the device, the purpose of the tube, and whether the tube is functioning as intended.[1,2] The insertion process can be aided by computed tomography, fluoroscopic illumination, endoscopic guidance, or during a surgical procedure.[1] For example, a tube or drain may be placed during a surgical procedure to promote drainage from collecting under the skin and prevent abscess formation, or an enteral feeding tube can be placed with endoscopic guidance to ensure correct placement and prevent early complications.[1,2] Developing an understanding of different percutaneous tube types, how they are used, and best practices in managing each type used is crucial to preventing and managing potential associated complications (Table 21-1).

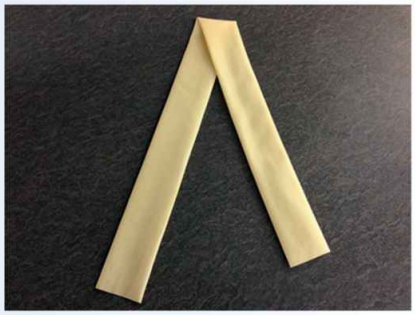
Table 21-1 Categories of Tubes and Drains

- **Simple tube/drain:** A catheter or a soft, flat, rubber drain. A rectal tube is an example of a simple tube that is inserted into the rectal cavity to relieve flatus, stool, or postoperative drainage. Simple tubes can be used for enema or medication administration.
- **Closed drainage catheter:** A catheter connected to a drainage container. In most cases, the collection container is considered sterile and sealed during the manufacturing process. Chest tubes, Jackson-Pratt drains, Hemovac drains, and indwelling urinary catheters (IUCs) are examples of closed drainage systems that attach to a bedside collection system.
- **Chest tube:** A sterile, closed drainage and suction system used in an emergency or surgical setting to relieve air or fluid from the pleural space. The closed drainage system is sterile and includes a water seal and negative pressure for suction.
- **Simple sump:** A catheter inserted into the abdominal cavity following surgery. It can be connected to a suction device at the distal end of an incision or can be used to instill an irrigant such as saline.
- **Nasogastric tube:** A tube that accomplishes nasogastric decompression when inserted through the mouth or nares into the stomach to remove gastric contents or administer food or medications.
- **Feeding tube:** A tube that is inserted into the stomach or small intestine for enteral feeding.
- **Indwelling urinary catheter:** A type of catheter that is inserted, using a sterile process, into the bladder to drain urine and decompress the bladder. A Coude catheter has a curved tip to ease insertion past the prostate.[1,2]

The use of percutaneous tubes and drains is increasingly common in healthcare and in many different care settings.[3–5] Using knowledge of anatomical location and reasons for the use of a tube will provide important management information.[2] In most instances, percutaneous tubes are named for the anatomical location being accessed by that tube and the specific product name. This provides the clinician important assessment considerations when setting up the plan of care. Important to safe and effective care is policies, procedures, and competencies that are based on the current literature and manufacturer guidelines.[1,2] Common tube types include tracheostomy, esophagostomy, biliary, nephrostomy, indwelling urinary, nasogastric, gastrostomy, and jejunostomy. Common to the successful management of most percutaneous tubes is stabilization to prevent erosion of the insertion site and subsequent leakage around the tube, accidental removal, and aspiration.[1–3]

A gap often exists in patient and family education regarding drains and tubes. Most people are aware of what to expect as far as an incision is concerned, but the presence of a tube or drain is unanticipated and unwelcomed. Preparing the patient for the potential outcome of a tube or drain placement reduces anxiety and gives the patient the opportunity to plan ahead (Table 21-2).

Table 21-2 Common Types of Tubes

Gastrostomy feeding tube	
Jackson-Pratt–style drain tube end	
Mushroom tip catheter style	
Penrose drain	
Sump style tube with sheath	

Patient Teaching

Tell the patient and family the location and the number of tubes and drains that might be needed after surgery in order to treat their medical condition.

Types of Tubes and Drains

Tracheostomy

A tracheostomy is a surgically created opening into the trachea, specifically at the anterior cervical location.[6] Common reasons for this procedure include situations obstructing the airway, long-term ventilator support, swallowing complications, and airway protection following trachea or laryngeal reconstruction surgery.[6,7] A tracheostomy tube is placed to maintain the stoma and airway. Because a tracheostomy is located between the upper airway and the lungs, it bypasses the normal process of transporting warm, moist oxygenated air and exhaling carbon dioxide and lung secretions.[6] Additionally, the larynx is bypassed preventing normal speech and communication.[6,7] Care and management of the person with a tracheostomy is critical to survival to that person. To promote a patent airway, patient communication, delivery of moist, warm air, and secretion control, the airway management plan should include involvement of speech therapy, respiratory therapy, and meticulous nursing care.[6,7] Developing policies and procedures for safe and effective care and patient education provide a standardized approach to care and fosters safe patient care.

A variety of tracheostomy tube sizes and shapes are available and are selected based on age and neck size. Some include an inner cannula. Due to added subcutaneous tissue lengthening the space between the skin level and the trachea, special consideration should be used for the person with higher body mass index. These individuals may require a larger stoma opening and special size and shaped tracheostomy tubes to reach the trachea. Tube stabilization, cleansing, and care are a high priority in the plan of care to prevent accidental extubation, aspiration, and infections.[6,7] Tracheostomy collars with a securing mechanism or tracheostomy ties are commercially available for this purpose and provide the stabilization need to maintain airway patency. Occasionally, wound care concerns will accompany standard tracheostomy care needs. Traumatic surgical procedures, postoperative infections, and, in some cases, the

need for a larger trachea opening can result in a wound around the trachea and added care concerns.[8] The use of absorbent, fenestrated dressings such as a foam product can provide local wound care combined with tracheostomy tube stabilization.[8,9] If the wound is complicated by infection, a silver-impregnated version of the dressing can be used to help treat the infection.[8,9] Dressing selection criteria include the amount of drainage or secretions compared to the absorbency features of the dressing, avoiding loose weave gauze that can release small fibers and become an aspiration risk, the size of the wound, and the condition of the wound bed.

Esophagostomy Tubes

An unusual tube to manage is the esophagostomy. As the name implies, this is a surgically created opening into the esophagus. The procedure involves surgical resection of the diseased segments, with the proximal end brought out through the skin to form a stoma. These stomas are flush with skin level and located lateral to the trachea, which leaves an uneven skin surface, potentially with creases, and affected by patient movement of the head and neck.[10] A potential complication of esophagostomy is the development of a fistula, which further complicates care.[11] These are among the more difficult pouching situations and often leak onto the peristomal skin. Consultation with a nurse specializing in the care of an ostomy (e.g., WOC nurse or enterostomal therapy nurse) provides an individualized assessment, combined with knowledge of product options for creative management solutions to contain drainage and provide patient comfort. Options include moldable solid skin barrier rings or sheets attached to a small pouching system. In cases of intractable nausea and vomiting, decompression tubes can be added.

Biliary Tubes

Situations causing obstruction of the liver bile ducts can be relieved by insertion of a biliary tube.[1,2] This tube is inserted into the liver and bile duct during either a surgical procedure or using fluoroscopy with contrast and guide wire. Catheter holes are positioned above and below the obstruction site. Drain tube options include cholecystectomy

tubes, percutaneous drains, T tubes, and endoscopically placed nasobiliary tubes. Tube stability, containing and quantifying the effluent or output, peritube skin care, and monitoring the output are the primary management concerns.[1,2] In some situations, drainage occurs through and around the tube causing difficulties in containment and skin integrity concerns due to the potential for moisture-associated skin damage.[2] The use of a urinary drainage bag with a tube adapter device to bring the biliary tube through the pouch will add stability of the tube and contain drainage off of skin to provide a healing environment. Patient education concerns include using a leg bag to facilitate gravity-assisted drainage and in some cases flushing the tube.[2]

Nephrostomy Tubes

Instances where there is obstruction at the renal pelvis of the kidney will prevent urine from flowing via the ureters to the bladder, leaving the affected person at risk for upper urinary tract damage.[2] To relieve this situation, nephrostomy tubes (temporary or permanent) are placed to facilitate urine drainage through the percutaneous tube and into an external containment system or pouch. The procedure is generally performed through interventional radiology using fluoroscopy with the patient under conscious sedation. The tube is placed percutaneously from the flank, through the kidney, and into the renal pelvis.[2] This procedure also provides for stent insertion and kidney access when stone removal or biopsy is necessary.[2] The proximal end of the tube has a pigtail end to secure the tube internally and external bumpers to stabilize at the insertion site. Tube stabilization is critical to prevent erosion of the insertion site with resulting urine leakage around the tube.[2] A variety of commercially made tube stabilization devices are available for this purpose.

Indwelling Urinary Catheters

Quality patient care is centered on preventing risk and complications that increase length of stay, prolong recovery periods, and increase the cost of care. The prolonged use of indwelling urinary catheters (IUCs) is one of these quality care concerns and is a frequent cause

of nosocomial infections in the acute care setting.[12,13] Normally a sterile environment, the bladder is exposed to external bacteria and other pathogens when a catheter remains in place.[12–14] Within a short time, a bladder infection can occur as the bladder is colonized, resulting in added discomfort and the potential need for antibiotics if catheter-related sepsis occurs.[12–14] Reasons IUCs are used include facilitating bladder emptying following surgical procedures where a recovery period is needed, containment of urine for those with sacral or coccygeal wounds where urinary incontinence is negatively impacting wound healing, and, in less desirable situations, managing urinary incontinence.[14] In most instances, IUCs should be removed as soon as possible to prevent urinary tract infections from occurring. In situations where urinary incontinence is a problem, referral to a continence nurse specialist or urologist provides opportunity to identify the cause, and set up a management plan based on that cause, and resolve or improve incontinence.[14]

A variety of IUCs are commercially available and include latex, silicone, and catheters coated with hydrogel, Teflon, or silver alloy.[14] Common properties include a double-lumen, balloon tip at the proximal end to secure into the bladder, a distal end to inflate the balloon and connect to bedside or leg drainage, and sufficient length to accommodate connection to a closed drainage system. Product selection is based on patient tolerance, expected length of time the IUC will be in place, and patient mobility. In most cases, the smallest size should be used to prevent trauma to the urethra mucosa and prevent other urologic complications.[14,15]

Management of IUCs begins with careful attention to sterile technique during insertion and when emptying the closed bedside or leg bag drainage system.[13–15] The longer the catheter is in place, the higher the risk for bacteria infiltration into the bladder and urinary tract infection.[14] While the catheter is in place, daily local care of the urinary meatus should be provided. This includes cleansing with soap and water.[12,14] Using a closed drainage system with limited disconnections of the tubing will also reduce bacterial migration into the bladder. Attention to catheter balloon size should also be considered.[14,15] Smaller balloon sizes (e.g., 5 mL) facilitate better

bladder emptying over large balloon sizes. Larger balloon sizes are generally reserved for use following prostatectomy to assist with hemostasis. The balloon stabilizes the catheter within the bladder.

Additional management concerns include monitoring for leakage around the catheter. This is an important assessment consideration as it could signal a kinked catheter, mucous blocking catheter opening, blood clots, or bladder spasms. Investigation into the cause of leakage should include a plan for correcting the cause of the problem and potentially removal of the catheter.[14] Assessing for and early identification of catheter-associated urinary tract infection (CAUTI) symptoms is another important aspect. Presenting symptoms include fever, chills, diaphoresis, hematuria, and pain. Unit-based competencies and strategies to care for IUCs provide a standardized approach to minimize catheter use, promote early removal, prevent and identify CAUTI, and promote urinary continence.[14,15]

Practice Point

Presenting symptoms of CAUTI include fever, chills, diaphoresis, hematuria, and pain (flank and suprapubic). Remove indwelling urinary catheters as soon as reasonably possible.

Enteral Feeding Tubes

Enteral feedings are critical to the nutritional support to the person debilitated by chronic illness, traumatic injury, complex surgical procedures, chronic and nonhealing wounds, or other conditions where malnourishment is a problem.[1–4] Its use does require an intact and functioning digestive system and is preferred over the use of parenteral nutrition.[1,2] Prolonged periods of inactivity in the small intestine can cause atrophy of the villi that lines the jejunum and ileum to absorb the smaller food molecules broken down higher in the

digestive tract.[1–4] Additionally, nutrients are better absorbed from the gastrointestinal track. These tubes can be placed by a variety of methods including surgical, endoscopic, radiological, or manually via the nares. Types include nasogastric, gastrostomy, and jejunostomy.[3,4]

Nasogastric Tubes

One of the most common and easy to place tube is nasogastric. It is the least stable of the enteral feeding options and carries the added risk for aspiration complications.[1] These tubes are inserted manually via the nares to the stomach, either during a procedure or at the bedside. Uses include stomach decompression, medication administration, or nutritional support. The patient receiving long-term enteral feeding from this method should be carefully evaluated for a less problem-prone option for nutritional support. Speech therapy to identify and manage swallowing concerns and registered dietitian services to determine specific nutritional support needs can provide important adjunct care.[1–4]

To reduce the chance of aspiration, the clinician inserting and managing nasogastric tubes should complete a competency that includes safe insertion technique and ongoing assessment of the patient for correct placement into the stomach.[16–18] The procedure involves first selecting the appropriate tube and measuring the anatomical markers to determine the correct length of the tube to be inserted. Once these are established, assemble the supplies, including the marked tube, water-based lubricant, a cup of water for responsive patients with an effective swallowing reflex to aid insertion, and any items specific to the product being used.[18] It is also important to prepare the patient and any family members on what to expect during and after insertion.[15] During insertion, maintain tube stability at the nares being used, and ensure the tube is well seated in the stomach prior to instilling fluids, medications, or formulas. X-ray placement verification of the tube location only determines correct positioning at the time the x-ray was taken. For this reason, a number of assessment steps should be taken to ensure correct placement and avoid instilling to the lungs. Options include ultrasound, visualizing the

tube, assessing the abdomen, and testing for the pH of a small gastric sample.[16–18] The use of stabilizing devices specific to nasogastric tubes is an important step in preventing accidental dislodgement of the tube out of the stomach into the lungs.[1,2]

Gastrostomy Tubes

A variety of insertion methods are used to place a gastrostomy tube, and the method used will depend on when the tube is placed (e.g., surgically or intervention radiology), the purpose of the tube (e.g., feeding or decompression with feeding), and condition of the patient. These tubes are inserted directly into the stomach and exit percutaneously at the anterior abdominal wall. In instances where stomach decompression with simultaneous jejunal feeding is used, a dual-chambered tube is used with one segment ending in the stomach and the other ending in the jejunum.[1,2,4]

Management concerns for gastrostomy tubes include tube stabilization, preventing aspiration, fluid management, preventing clogs, medication interaction, and nutritional support.[19] The registered dietitian is an important team member for these patients and will assess for protein–calorie and energy needs based on the disease process, select the appropriate formula, and determine appropriate calorie volume and fluid needs.[1,2]

Percutaneous Endoscopic Gastrostomy Tube

Percutaneous endoscopic gastrostomy (PEG) tubes are inserted directly into and through the abdominal wall using an endoscope.[1,2,19] The tube exits proximally at the abdominal wall. Its purpose is to treat and prevent nutritional deficits associated with chronic disease complications, following extensive and complicated surgery, and to support the healing process of chronic wounds.[19] Stabilization of the tube occurs with an internal bumper against the stomach wall and external bumper placed cutaneously along the external abdominal wall.[1,2] Most tubes manufactured to be specific to gastrostomy feeding include this internal and external bumper. For those tubes without this

support, commercially manufactured devices are available to stabilize the tube.

Enteral Tube Feedings

Common problems associated with enteral feeding tubes include leakage of gastric contents around the tube onto the peritube skin, moisture-associated skin damage, and clogged tubes. Since gastrostomy tubes are placed into the stomach, peristalsis can cause unstabilized tubes to migrate and erode and enlarge the fistula.[1,2,19] Stabilizing the tube, either through the stabilizing device included with the product or a commercially manufactured device, will prevent this problem. In situations where leakage around the tube has occurred, stabilization will prevent tube migration, allowing the fistula to contract around the existing tube.[1,2]

Tube clogging or obstruction in enteral feeding tubes is generally caused by formula exposure to medications, medication interaction, limited water flushes, or use of tubes not intended for enteral feeding use. In most instances, use of water flushed before and after each medication administration and formula reduces the chance of tube obstruction.[1,2] Whenever possible, use of liquid over crushing pills is a useful strategy. Time-released medications should not be used with enteral tubes, and pharmacist consultation can provide suitable medication alternatives.[1,2] Addressing hydration needs through regular water flushes (based on individual hydration needs) facilitates tube cleansing. In the event of an obstructed tube, instilling warm water along with gentle agitation with the syringe can help loosen the obstruction. For intractable obstructions, the use of pancreatic enzyme–based clog busters can dissolve the material obstructing the tube.[1,2] Finally, having a matching extra enteral feeding tube on hand provides backup if the tube clog is not amenable to these strategies. In some situations (e.g., infants, young children, and dementia-related conditions), accidental tube removal can occur when the patient pulls on the tube.[19] This is another indication to have the extra tube on hand. Strategies to prevent accidental tube removal include stabilizing devices, use of garments to cover the tube site, and use of low-profile tubes that are much harder to pull on.

Jejunostomy Tubes

Some patients with long-term enteral feeding needs will have a jejunostomy tube placed. This tube is surgically or endoscopically placed and inserted directly into the jejunum.[1,4] Reasons for selecting this type of tube include those at high risk for aspiration or those with esophageal, gastric, or duodenal disease process where a gastrostomy tube is not feasible.[1,4] Tube stability is achieved with internal and external bumpers.[1,2]

Practice Point

Medications that can be administered in liquid form are best suited for enteral feeding tubes. Water flushes between medications and feedings will help prevent tube obstruction.

Always determine if it is safe for the nurse, as well as the patient, to crush a medication in pill form. Crushing some medications can expose the nurse to harm. Crushing a pill may render it unsuitable for administration via the enteral route.

Management of Tubes and Drains

Peritube skin care includes daily assessment for skin integrity concerns, signs of infection, leakage around the tube, and the potential for moisture-associated skin damage.[1,2,19] A dry fenestrated dressing in the first few days following insertion will absorb procedure-related draining (Table 21-3).[1,2] Once the site is healed, care includes daily cleansing with water and monitoring for problems. Manufacture guidelines should be followed for the care of individual tubes, and policies and procedures should be in place addressing tube care needs. Documentation should include the specific product used, care and patient response, condition of the tube and peritube skin, and interventions used to manage or follow up on complications.[1,2,19]

Table 21-3 Nursing Care of Feeding Tubes

Successful nursing care of feeding tubes depends on:

- ensuring that the device is stable
- providing appropriate nutritional management
- meeting the patient's hydration needs
- using the proper technique for instillation of medications.

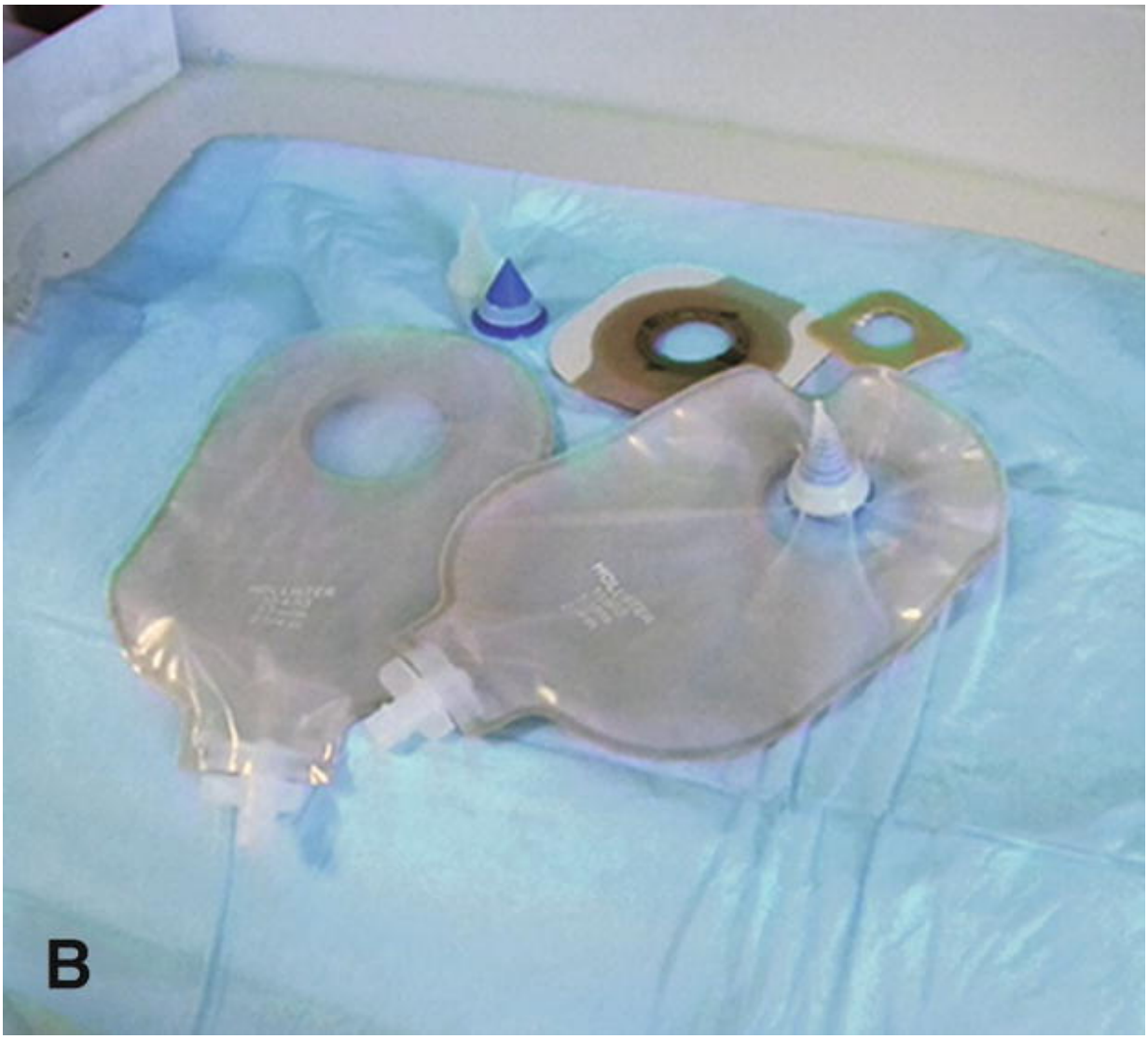
A holistic assessment provides individualized information to develop management plans that prevent complications, use of appropriate equipment, and early identification of problems. Using knowledge of the anatomy and physiology of involved body systems combined with the rationale for use of a tube or drain provides important information in specific care concerns, obtaining needed product, injury prevention, and documentation considerations.[1,2,19] Depending on the care setting, patient and family/caregiver education, addressing socioeconomic and psychosocial concerns, and involving the interdisciplinary team provide a comprehensive approach to care. Interdisciplinary team members include the pharmacist, registered dietitian, speech therapy, wound and ostomy care nurse (or enterostomal therapy nurse), and practitioner placing the tube.[1,2,20] Common care concerns include stabilizing the tube, appropriate product use, preventing complications such as infection, and nutritional support.[1,2,19,20] Discharge planning for long-term tubes should address safety, handoff communication needs, specific product and management strategies, and another care needs specific to this patient. The Patient Scenario discusses the nursing management of a patient with multiple drains.

PATIENT SCENARIO

Nursing Management of Multiple Drains

A patient had two abdominal drains due to pancreatitis and a colonic leak. Secondary to massive sepsis, the drain sites started to leak large amounts of fluid around the drains creating a containment and nursing management problem.

As seen in the first photo (Fig. 21-1A), the abdomen has two drain tubes; the exit sites have marked erythema, erosion, and hydrated tissue at the drain sites. Because the two drains are in close proximity, creating a reliable and sustainable containment system is challenging.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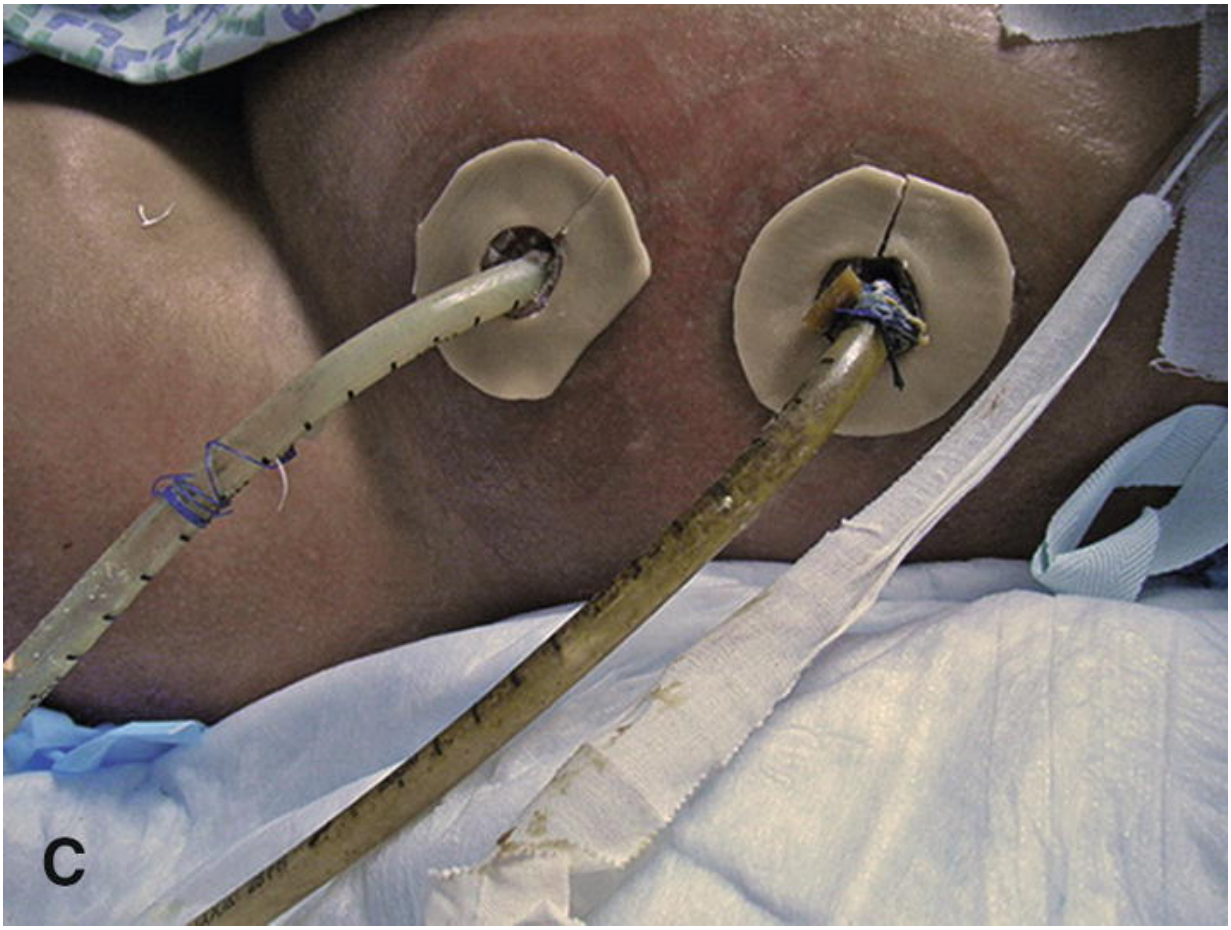
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Figure 21-1. (A) Initial presentation of patient with two abdominal drains in place. **(B)** Pouching supplies. **(C)** Moldable skin barrier rings around the tubes. **(D)** Faceplate of the two-piece pouching system in place around the tubes. **(E)** Applying the tube access port. **(F)** Pouch applied to faceplate. **(G)** Both pouches secured.

Nursing goals to manage the drains included:

1. Stabilize the tubes to prevent further erosion of the abdominal tube sites.
2. Contain the drainage through a reliable pouch seal while providing access for the tubes through the pouching system.

3. Quantify the drainage.
4. Promote optimal environment for wound healing.

Tube Management

To both stabilize the drains and maintain a reliable pouching system, WOC nursing recommendations included the use of a two-piece pouch with a carboxymethylcellulose (CMC) skin barrier, gelatin-based moldable rings with tack on both sides, and a tube access port (Fig. 21-1B).

The pouch, rings, and paste were applied as follows:

The pouch was prepared with the addition of a tube access port through the anterior side of the pouch.

The gelatin-based skin barrier moldable ring was applied around the first drain tube (Fig. 21-1C) and secured with the faceplate of the two-piece pouching system to provide a reliable seal to the liquid drainage leaking around the tube (Fig. 21-1D).

With the help of hemostats and a water-soluble lubricant, the drain was pulled through the drain tube access port to facilitate tube drainage (Fig. 21-1E). The pouch could then be coupled to the faceplate (Fig. 21-1F).

The second drain tube (not leaking) was managed with the gelatin-based skin barrier ring and faceplate with tape collar trimmed to fit close the first drain tube (Fig. 21-1G).

The pouch end was closed and allowed to drain by gravity. The spout end of the pouch allowed for ease of emptying and quantifying the drainage.

Source: Photos and case study provided courtesy of Linda Stricker and Amelia Agostinelli.

Patient Teaching

Explain to patient and family the importance of tube stabilization in preventing moisture associated skin damage (MASD).

Fistula Types

Enterocutaneous Fistula

The development of an enterocutaneous fistula (ECF) is devastating to the patient in terms of quality of life and potential for life-threatening complications and an interdisciplinary care team challenge.[21–23] A simple definition of an ECF is the abnormal opening or connection between two body organs. Occurrences can happen between two internal organs or from an internal organ to the skin surface leading to uncontrolled drainage from the body organ onto the epithelium.[21,22] The basis for naming and assessing any specific fistula is the physiologic origin and point of exit.[23]

ECFs are an uncommon but serious complication to a disease process or surgical procedure.[23,24] Risk factors for spontaneous ECF development include inflammatory bowel disease such as Crohn's disease, history of radiation therapy, and cancer.[23,24] Risk factors for ECF development in the surgical patient include inadequate blood supply at the surgical site, insufficient suturing technique, traumatic situations (e.g., lacerating wound to the abdomen) leading to incomplete bowel prep, and nutritional deficit prior to surgery.[21–23,25] Management considerations to minimize these risk factors include an interdisciplinary team approach to disease management, preoperative nutritional support, adequate intravenous fluid support, and, in some situations, the use of prophylactic broad-spectrum antibiotics.[20–23,25] In cases where the surgical site is considered to be a poor healing risk, the use of secondary and tertiary wound healing is used, or in rare cases, a temporary diverting ostomy is created above the anastomotic site while this area heals.[21]

Mortality rates for patients with an ECF range from 5.5% to 30%, with infection, fluid and electrolyte imbalance, and poor nutrition as the most common factors leading to death.[21–23] In some instances, with early detection and supportive care, fistulas will close spontaneously within 4 to 5 weeks; however, fistulas not closing spontaneously are likely to require surgical closure once the inflammatory process, infection, fluid balance, and nutritional concerns have been stabilized.[21–23] The presence of an ECF prolongs the recovery period

and adds significant costs to care. Depending on the insurance coverage, the supply costs may or may not be covered. Since management includes the use of more complex products to prevent skin erosion, and contains the effluent, the issue of who is paying for these supplies adds stress to the plan of care. Additional costs, sometimes as much as $270,000, are incurred from diagnostic tests, nursing and medical care, nutritional support, and surgical intervention.[26] Advances in nutritional support options, containment devices, and fluid and electrolyte maintenance contribute to patient recovery and positive outcomes (Table 21-4).[21–23,25]

Table 21-4 Types of Fistulas

Fistula	Connection
Colocutaneous	Colon to skin
Rectovaginal	Rectum to vagina
Entero-entero	Small intestine to small intestine
Enterocutaneous	Small intestine to skin

Classification of ECFs

There are several classification methods used to describe a fistula. This includes the type and amount of effluent present, anatomy involved, and complexity of the situation. Classifying the fistula using these methods provides important assessment, management, and documentation information for the individualized plan of care.[21,22] High-output fistulas require additional nutrition and fluid support, and containment strategies, while low-output fistulas may only require absorbent dressings with nutrition support.[21–24] More complicated fistulas are associated with abscesses and multiple organs, and the fistulas open in the base of a wound, also called atmospheric fistula, represent additional involvement of healthcare providers.[21–24]

The amount of effluent is an important assessment consideration. Low output is defined as less than 200 mL in a 24-hour period and 200 to 500 mL for moderately draining fistula, and high-output fistulas will drain more than 500 mL or more in a 24-hour period.[21,22,26–28] Precise monitoring and measuring of the output is crucial to managing fluid balance.[21,22,24] In general, the more proximal the fistula location, the higher the anticipated output. Additional clues to assess for include color and consistency. Gastric drainage is clear to light yellow-green with a watery consistency, with a pH of about 3.0. Biliary drainage is viscous with gold to deep green color and a pH of 7.5. With a pH of 8.3, pancreatic drainage will present as clear and watery. Considering the normal pH of the skin is 4.5 to 5.5, pH differences need to be considered with a goal to protect the adjacent skin from moisture- and chemical-associated injury.[21,22]

Goals of Management

Managing the person with an ECF is a complex process requiring diagnostic testing to identify the cause and location of the fistula and the creative skills and attention from the interdisciplinary team.[21–23] In addition to the surgeon and licensed independent practitioner, the wound and ostomy care nurse (or enterostomal therapy nurse) with knowledge of product and containment options, the registered dietitian with knowledge of fluids and nutritional needs, and the social services to facilitate psychosocial support are among the crucial team members in providing care.[20,25] Those with long-term care needs are likely to require multiple care settings such as home care, long-term care, ambulatory, and acute care during the timeline to ECF closure.[21–24,29]

Management goals begin with knowledge of risk factors leading to ECF formation, early identification of impending or presenting fistula, and coordinating healthcare providers in managing nutritional, hydration, containment, and infection complications.[21–23,25] Following abdominal surgery, or other patients at risk, fever, localized edema, indurated skin, increased pain, changes in fluid and electrolyte balance, and altered mental state should be carefully monitored and reported to the surgeon or licensed independent practitioner for additional care.[23,24,28,29]

Once the presence of an ECF is suspected, additional diagnostic tests should be performed to determine the nature and extent of the fistula, specifically, to identify the beginning and end point. Fistulograms, positron emission tomography (PET) scans, and magnetic resonance imaging (MRI) provide visibility of the fistula in more detail.[21–23,28] Determining the full extent of the ECF and what supportive care may be needed provides critical management guidance. ECF drainage will include sodium, potassium, magnesium, zinc, proteins, digestive enzymes, and fluid, all which directly contribute to the nutrition deficit and electrolyte depletion that will result if left unmanaged.[25] Uncontained drainage contains pH differences and enzymatic destruction of the skin, which exposes the

dermis, rendering the normally protective layer of the epidermis less effective in protecting from pathogen invasion and increased pain as nerve endings are exposed.[21–23] In some situations, it will be necessary to initially withhold oral intake while the full extent and nature of the ECF has been identified and a management plan is established.[21–23,25,28]

Medical Management

Medical management goals should address four primary concerns: fluid and electrolyte balance, nutritional support, infection prevention and management, and diagnosing the exact location and extent of the fistula.[21–23,25,28] Early identification and treatment with these goals in mind will optimize and stabilize patient condition and increase the chance of spontaneous fistula closure. While only a third of these cases will achieve spontaneous closure, it is still the desired outcome. In most instances, oral fluids and feedings are initially held with nutritional support provided through total parenteral nutrition.[23,25] Resuming oral feedings or initiating enteral feedings should be considered to prevent mucosal villous atrophy in the jejunum and ileum and promote optimal nutrient absorption.[21,22,25] Total parenteral nutrition, however, does remain a viable option when oral or enteral feeding is not possible.[21] Continued monitoring of blood glucose levels, serum electrolytes, and patient weight provides important clues to the effectiveness of the nutritional plan of care.[23,25] There are some pharmacologic options that can be attempted to reduce output from a high-volume fistula (Table 21-5). Octreotide or somatostatin is sometimes used for this purpose; however, results will vary and are unpredictable.[21,23,30] If improvement is not seen within 2 to 3 weeks, the medication should be discontinued. Continuous vigilance in monitoring for signs of sepsis is crucial as mortality rates from this complication increase in the presence of infection.[21–23]

Table 21-5 Pharmacologic Options in Fistula Management

Classifications	Examples	Effect on Effluent
Antimotility drugs	Loperamide, codeine, tincture of opium	Slows intestinal motility and allows more nutrient absorption time
Antisecretory drugs	Proton pump inhibitors, histamine-2 receptor agonists, octreotide, somatostatin	Slows gastric secretions and slows intestinal motility
Supplements	Bile salts, pancreatic enzymes	Improve absorption of lipids and proteins

From Tappenden, K.A., Quatrara, B., Parkhurst, M.L., et al. Critical Role of Nutrition in Improving Quality of Care. *Journal of Parenteral and Enteral Nutrition* 37(4):482-97, 2013; Nix, D., Bryant, R. Fistula Management. In J.E. Carmel, J.C. Colwell, M.T. Goldberg, eds. *WOCN® Core Curriculum: Ostomy Management*. Philadelphia, PA: Wolters-Kluwer, 2016:201-19.

Spontaneous ECF closure is sometimes achieved within 6 to 8 weeks with rigorous interdisciplinary management.[23,31,32] Plans for surgical closure will likely need to be considered when spontaneous closure does not occur. Risk factors preventing spontaneous closure include sepsis, protein–calorie malnutrition, a distal obstruction, a matured fistula (or epithelialized fistula tract), exposed mucosa, and inability to reduce high-output effluent.[25,32] There has been some attention to the use of a combination of fibrinogen and thrombin to create a biological glue to close some fistulas.[33] Best practices for this procedure include initial debridement of devitalized tissue, saline irrigation, and then injection of the fibrin glue product directly into the fistula. It is likely that more than one injection will be needed to achieve the intended result of coagulation and closure of the fistula tract.[33]

Surgical closure of the fistula is planned when medical management fails. Successful surgical outcomes are more likely achieved through careful planning, fluid and electrolyte and nutritional optimization, and resolution of infection and inflammation.[32–34] This also reduces the incidence of peritonitis and sepsis or developing a new fistula.[32,35] Timing and surgical approach are determined by the anatomical details of the fistula and patient readiness.[32] For those who are not stable enough for surgery, medical management will continue.[21]

Practice Point

Principles of enterocutaneous fistula care include careful attention to nutrition options, hydration needs, odor and drainage containment, skin care, and managing infection.

Nursing Management

Nursing care and management for these patients involve a holistic approach to assessment and intervention. A review of the medical record for a history of the problem and the current situation and results of diagnostic tests provide important information on what should be monitored.[21,22] Considerations should include a physical assessment of the affected area, including skin integrity, and the psychosocial and discharge planning needs. Particular attention to the amount and nature of fistula effluent, skin integrity of the perifistula area, infection prevention and early identification, and containment needs forms the basis of the nursing plan.[21,22] Skin care is of primary concern due to the potential for skin damage that can increase infection risk and interfere with the integrity of containment products.[21–23]

The wound and ostomy nurse (or enterostomal therapy nurse) is a valuable resource in the nursing care of these patients because of the knowledge of different pouching products and how they are used and how the abdominal skin contours will affect pouching or containment application and reliability.[21,22,36] Specific care concerns include precise measurement of effluent, odor control, perifistula skin management, cost containment, and education to patients and those involved in care. Product options to aid in containing effluent include ostomy pouches, solid skin barriers, wound care pouches and accessories, catheter holders, tube access devices, odor-controlling agents, and suction.[21,22] In many cases, it will be combination of

product and changes in the plan based on patient response that provide optimal and cost-effective care. Low-output ECFs (<100 mL/d) can be managed with absorbent dressings with charcoal options to manage odor when needed.[21,22,36]

Practice Point

Even the most skilled nurse will be challenged in pouching a wound with an ECF, but an individualized approach, assessment of skin contours, creativity, and persistence will lead to a predictable system that meets management goals.

Use of Pouching

The goals of using a pouching system to manage a fistula include drainage containment, protecting and healing perifistula skin, and providing for moist wound healing.[21,22,36] High-output ECFs, and those located at the base of an abdominal wound, present complex containment problems. Many times, more than one person will be needed to apply the pouch. The high output combined with a large area and inconsistent skin contours that are flush with the wound margins are difficult to manage and achieve a predictable and reliable pouch seal.[36] The use of moldable solid skin barriers, skin barrier paste and powder, and an appropriate-sized pouch will facilitate containment and drainage measurement.[21,36] Assess the skin contours and wound measurements with the patient in a supine and sitting position, so any changes in the skin are exposed and taken into consideration for the pouching product(s).[36] Patient mobility is another concern when applying a pouching system, and by first assessing these skin contours in different patient positions, the nurse can visualize and address where leakage could occur.[21,22,36] Additional skin assessment considerations include observing for scars and

creases, which can also weaken the pouch seal.[36] The use of skin barrier paste and moldable solid skin barriers cut and timed to a wedge shape will even out skin contours, fill creases, and promote a level pouching surface.[36] To provide more flexibility in the seal, cut radial slits (darts) around the skin barrier opening of the pouch prior to pouch application.[21,22] Sizing of the pouch and any skin barrier wedges used should be as close to the wound margins as possible, and any exposed skin can be covered with barrier ointments once the system is in place.[21,22,36] Wound pouches with windows will facilitate this process.

Even the most skilled nurse will be challenged in pouching these wounds.[34] Careful assessment and an individualized approach combined with reassessment and persistence will lead to a predictable system. Once a successful pouching system is achieved, providing step-by-step instructions, product lists, and a pattern is useful for those who are providing care. Achieving a reliable system adds to cost containment as supplies can then be budgeted.[21,26] Documenting the assessment, including the details of the skin and containment needs, the specific product used, and the wear time, provides a historical record and useful information in situations where the insurance policy will cover these supplies.

Negative Pressure Wound Therapy

Some fistulas can be managed through the use of negative pressure wound therapy (NPWT), including a combination of the NPWT product to facilitate healing, with the fistula being isolated from the wound, and a pouching system applied and NPWT simultaneously used to manage the wound. NPWT involves the use of a suction device combined with a sponge or gauze in the wound bed covered with a transparent adhesive dressing.[22,37,38] NPWT reduces edema and drainage in the wound bed to support the healing process.[37] Indications for use of NPWT with fistula care include a high potential for spontaneous closure (specifically those not matured), no evidence of exposed bowel or other internal organ, and no abscess formation complicating the situation.[38] Careful attention to safety with NPWT is a primary consideration as the addition of suction could induce new

fistula formation. For this reason, a contact layer such as silicone- or petrolatum-based product should be considered as an interface between the wound and the suction device.[37,38] Manufacturer guidelines should be followed when using these devices in order to prevent further damage and promote best outcomes.[37] Nurses applying NPWT devices should be deemed competent in the use of these products.

Case Management

The costs to manage ECFs are high. In many situations, there is limited insurance coverage for the assortment of product needed to successfully care for these patients.[26] Additionally, different levels of care are likely to be involved. The psychosocial and emotional toll on well-being can be enormous with dramatic quality of care concerns. Case management and discharge planning specialists should be involved as soon as possible to promote communication and determine best options for care.[26,29] Considerations when placing or moving the patient to a different care setting include whether the pouching system is reliable for at least 24 hours, if NPWT is being successfully used, the skills and abilities of subsequent care providers, education needs, obtaining products for care, and insurance coverage.[21,37,38]

The development of an ECF is devastating for patients and a complex management problem for the interdisciplinary team. These individuals are at high risk for developing comorbid conditions including sepsis, malnutrition, fluid and electrolyte depletion, containment and odor control, and skin damage.[21,22] The mortality associated with ECF and the potential complications demonstrate the need for early identification and patient support that begins with assessing for ECF risk factors and providing care that mitigates these risks and recognizing the signs and symptoms of ECF development. Goals of care are centered on stabilizing nutrition and fluid and electrolytes, containing and monitoring effluent, managing odor, preventing and treating infection, and using an interdisciplinary approach to care.[22,23,25] There are a variety of wound and pouching

options to support management with NPWT as an option under certain conditions.

Summary

Successful management of tubes and drains involves an understanding of the specific product being used, knowing the anatomical structures involved, the rationale for the tube or drain, and specific management concerns. Stabilizing the tube and drain patency as well as assessing and protecting the peritube skin is imperative and forms the basis of the management plan.[1,2] Classification of fistulas includes the anatomical beginning and end point and the nature and amount of effluent.[23] Fistulas are named for the anatomical beginning and end points that provides additional information for management concerns. Effluent concerns, such as volume and odor, should be carefully assessed along with the potential for skin damage and containment options used to address these problems. Consideration and planning for hydration, electrolyte balance, and nutritional support are complex and primary concerns that are best approached through the skills and communication of the interdisciplinary team.[21–23,25]

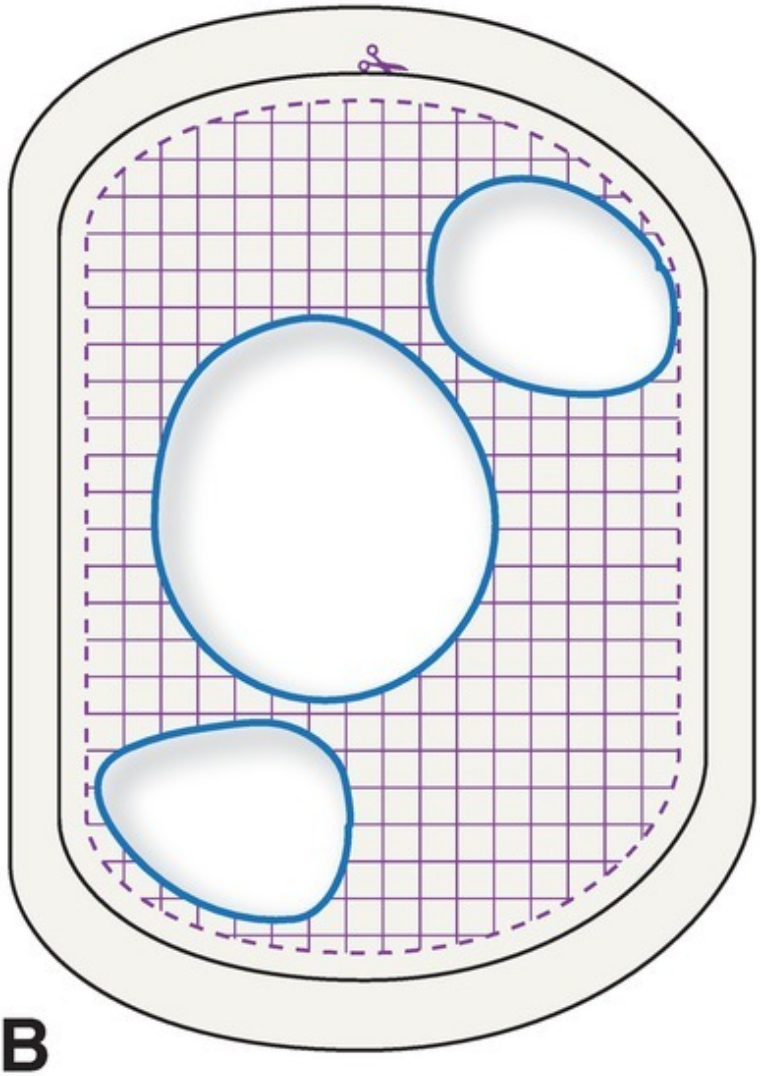
PATIENT SCENARIO

Clinical Data

A 64-year-old patient with colon cancer and metastasis is admitted for management. Despite multiple surgeries, chemotherapy, and radiation, the disease process progressed. Other problems include cachexia, inoperable colorectal mass partially obstructing effluent, colostomy, and anemia. The situation was complicated by a perforated viscus and resulting peritonitis resulting in four abdominal fistulas on the abdomen.

Case Discussion

Abdominal assessment demonstrated marked erythema in the perifistula skin and uneven skin contours making pouching considerations difficult (see Fig. 21-2A).



B

Figure 21-2. (A) Initial assessment of patient's abdomen reveals multiple fistulas with erythema around the perifistula skin. Figure **B** shows the template that the nurse created to be used to cut out the skin barrier of the wound manager pouching system. **(C)** Wound manager pouching system in place on the patient's abdomen.

Pouching Techniques

Initial pouching attempts included the use of soft convexity, which leaked within 15 hours. The final solution to manage the situation included the use of moldable skin barrier seals to even the abdominal contours. To promote a more secure seal, skin cement was used combined with the skin barrier seals. A wound manager pouching system was used with multiple openings cut away for each fistula (see Fig. 21-2B) which illustrates the template made to accommodate the openings for the fistulas on the patient's abdomen. Skin barrier paste was used to promote a secure seal with the paste applied directly around the fistulasopenings. The moldable skin barrier seals with the pouch was then applied over this (see Fig. 21-2C).

Conclusion

This yielded a 48-hour seal and promoted patient comfort, drainage and odor containment, and improved skin condition.

Source: Photos and case study provided courtesy of Sandra Hughes, BSN, RN, CWOCN.

Show What You Know

1. **Which of the following is a definitive indicator that a fistula has developed in an abdominal surgical wound?**
   A. Fever and erythema at the wound bed
   B. Wound drainage and dehiscence
   C. Gastrointestinal contents in the wound bed
   D. Abdominal pain and serosanguineous exudate

2. **Optimal functioning of enteral feeding tubes depends on which primary nursing considerations?**
   A. Hydration, location of tube, and diagnosis
   B. Skin care, hydration, location of tube
   C. Tube stabilization, hydration, nutrition
   D. Nutrition, location of tube, continuum of care

3. **Which of the following conditions predispose a patient to formation of an enterocutaneous fistula?**
   A. Normal nitrogen balance
   B. Albumin level of 4.0 g/dL
   C. Blood glucose of 80 mg/dL
   D. Crohn's disease

4. **A patient with an enterocutaneous fistula is averaging an output of 9,600 mL over a 24-hour period. The direct care staff is concerned about pouch overflow and leakage. What would you advise?**
   A. Discontinue the pouch and switch to gauze dressings.
   B. Connect the pouch to bedside gravity drainage.
   C. Add more skin barrier paste and tape to caulk the leaks.
   D. Restrict enteral and intravenous fluids.

Answers to these questions are found on page 687.

References

1. Bryant, R.A. Percutaneous Tube Management. In Bryant, R.A., Nix, D.P., eds. Acute and Chronic Wounds: Current Management Concepts, 5th ed. St. Louis, MO: Elsevier, 2016:562-78.
2. Fellows, J., Rice, M.C. Nursing Management of the Patient with Percutaneous Tubes. In D.B. Doughty, L.L. McNichol, eds. WOCN® Core Curriculum: Wound Management. Philadelphia, PA: Wolters Kluwer, 2016:723-31.
3. Hartwell, J.L., Cotton, A., Rozycki, G. Optimizing Nutrition for the Surgical Patient: An Evidenced Based Update to Dispel Five Common Myths in Surgical Nutrition Care. American Surgeon 84(6):831, 2018. Available at: https://search-ebscohost-com.contentproxy.phoenix.edu/login.aspx?direct=true&AuthType=shib&db=f5h&AN=130668627&site=ehost-live&scope=site. Last accessed May 12, 2019.
4. Speer, E.A., Chow, S.C., Dunst, C.M., et al. Clinical Burden of Laparoscopic Feeding Jejunostomy Tubes. Journal of Gastrointestinal Surgery 20(5):970-5, 2016. doi:10.1007/s11605-016-3094-2.
5. Chen, Z., Su-yong, L., Qi-bao, D., et al. The Effects of Pre-operative Enteral Nutrition From Nasal Feeding Tubes on Gastric Outlet Obstruction. Nutrients 9(4):373, 2017. Available at: https://search-proquest-com.contentproxy.phoenix.edu/docview/1899818976?accountid=35812. doi: http://dx.doi.org.contentproxy.phoenix.edu/10.3390/nu9040373.
6. Myatt, R. Nursing Care of Patients with a Temporary Tracheostomy. Nursing Standard 29(26):42, 2015. Available at: https://search-proquest-com.contentproxy.phoenix.edu/docview/1855438065?accountid=35812. doi: http://dx.doi.org.contentproxy.phoenix.edu/10.7748/ns.29.26.42.e9742.
7. Sole, M.L., Talbert, S., Aragon Penoyer, D., et al. Characteristics, Resource Utilization, and Nursing Care of Patients Who Undergo Percutaneous Tracheostomy. Clinical Nurse Specialist 28(5):288-95, 2014. doi:10.1097/NUR.0000000000000072.
8. Hartzell, L.D., Havens, T.N., Odom, B.H., et al. Enhanced Tracheostomy Wound Healing Using Maltodextrin and Silver Alginate Compounds in Pediatrics: A Pilot Study. Respiratory Care 59(12):1857-62, 2014. doi:10.4187/respcare.02822.
9. Wen-Ling, C., Wen-Pei, H., Mei-Hsing, C., et al. Gauze vs. Solid Skin Barrier for Tracheostomy Care: A Crossover Randomized Clinical Trial. Journal of Wound, Ostomy, and Continence Nursing 40(6):573-9, 2013.
10. MacKenzie, D.J., Popplewell, P.K., Billingsley, K.G. Care of Patients After Esophagectomy. (Cover Story). Critical Care Nurse 24(1):16-31, 2004. Available at: https://search-ebscohost-com.contentproxy.phoenix.edu/login.aspx?

direct=true&AuthType=shib&db=a9h&AN=12598418&site=ehost-live&scope=site. Last accessed May 12, 2019.
11. Rahmi, G., Perretta, S., Pidial, L., et al. A Newly Designed Enterocutaneous Esophageal Fistula Model in the Pig. Surgical Innovation 23(3):221-8, 2016.
12. Gesmundo, M. Enhancing Nurses' Knowledge on Catheter-Associated Urinary Tract Infection (Cauti) Prevention. Kai Tiaki Nursing Research 7(1):32-40, 2016. Available at: https://search-ebscohost-com.contentproxy.phoenix.edu/login.aspx?direct=true&AuthType=shib&db=ccm&AN=119345448&site=ehost-live&scope=site. Last accessed May 13, 2019.
13. Parker, V., Giles, M., Graham, L., et al. Avoiding Inappropriate Urinary Catheter Use and Catheter-Associated Urinary Tract Infection (CAUTI): A Pre-post Control Intervention Study. BMC Health Services Research 17(1):314, 2017. doi: 10.1186/s12913-017-2268-2.
14. Moore, K.N., Franklin, L. Indwelling & Intermittent Catheterization. In D.B. Doughty, K.N. Moore, eds. WOCN® Core Curriculum: Continence Management. Philadelphia, PA: Wolters-Kluwer, 2016:232-49.
15. Feneley, R.C., Hopley, I.B., Wells, P.N. Urinary Catheters: History, Current Status, Adverse Events and Research Agenda [Published Correction Appears in J Med Eng Technol. 2016;40(2):59]. Journal of Medical Engineering and Technology 39(8):459-70, 2015. doi: 10.3109/03091902.2015.1085600.
16. Kim, H.M., So, B.H., Jeong, W.J., et al. The Effectiveness of Ultrasonography in Verifying the Placement of a Nasogastric Tube in Patients with Low Consciousness at an Emergency Center. Scandinavian Journal of Trauma, Resuscitation and Emergency Medicine 20:38, 2012. doi: 10.1186/1757-7241-20-38.
17. Williams, T. Nasogastric Tube Feeding: A Safe Option for Patients? British Journal of Community Nursing 21(Suppl 7):S28-31, 2016. doi: 10.12968/bjcn.2016.21.Sup7.S28.
18. Rushing, J. Inserting a Nasogastric Tube. Nursing 35(5):22, 2005.
19. Rahnemai-Azar, A.A., Rahnemaiazar, A.A., Naghshizadian, R., et al. Percutaneous Endoscopic Gastrostomy: Indications, Technique, Complications and Management. World Journal of Gastroenterology 20(24):7739-51, 2014. doi: 10.3748/wjg.v20.i24.7739.
20. Tappenden, K.A., Quatrara, B., Parkhurst, M.L., et al. Critical Role of Nutrition in Improving Quality of Care. Journal of Parenteral and Enteral Nutrition 37(4):482-97, 2013.
21. Nix, D., Bryant, R. Fistula Management. In J.E. Carmel, J.C. Colwell, M.T. Goldberg, eds. WOCN® Core Curriculum: Ostomy Management. Philadelphia, PA: Wolters-Kluwer, 2016:201-19.
22. Bryant, R.A., Best, M. Management of Draining Wounds and Fistulas. In R.A. Bryant, D.P. Nix, eds. Acute and Chronic Wounds: Current Management Concepts, 5th ed. St. Louis, MO: Elsevier, 2016: 541-61.

23. Whelan, J.F., Ivatury, R.R. Enterocutaneous Fistula: An Overview. European Journal of Trauma and Emergency Surgery 37:251-8, 2011.
24. Harle, K., Lindgren, M., Hallbrook, O. Experience of Living with an Enterocutaneous Fistula. Journal of Clinical Nursing 24:2175-83, 2015.
25. Badrasawi, M., Shahar, S., Sagap, I. Nutritional Management in Enterocutaneous Fistula: What is the Evidence? The Malaysian Journal of Medical Science 22(4):6-16, 2015.
26. O'Shea, J. Enterocutaneous Fistula: What Is the Real Cost of Care? Journal of Stomal Therapy Australia 33(1):26-30, 2013.
27. Sapunar, L.C., Sekovski, B., Matic, D., et al. Percutaneous Embolization of Persistent Low-Output Enterocutaneous Fistula. European Radiology 22:1991-7, 2012.
28. Tong, C.Y., Lim, L.L., Brody, A.R. High Output Enterocutaneous Fistula: A Literature Review and a Case Study. Asia Pacific Journal of Clinical Nutrition 21(3):464-9, 2012.
29. Slater, R.C. Supporting Patients with Enterocutaneous Fistula: From Hospital to Home. British Journal of Community Nursing 33(1):26-30, 2011.
30. Carrera-Guermeur, N., Martin-Crespo, R.M., Ramirez, H.J., et al. Octreotide & Enterocutaneous Fistula Closure in Neonates & Children. European Journal of Pediatrics 175:305-12, 2016.
31. Latifi, R., Gustafson, M. Abdominal Wall Reconstruction in Patients with Enterocutaneous Fistula. European Journal of Trauma and Emergency Surgery 37:241-50, 2011.
32. Heimroth, M.S., Chen, E., Sutton, E. Management Approaches for Enterocutaneous Fistula. The American Surgeon 84:326-33, 2018.
33. Wu, X., Ren, J., Wang, G., et al. Evaluating the Use of Fibrin Glue for Sealing Low-Output Enterocutaneous Fistulas: Study Protocol for a Randomized Controlled Trial. Trials 16:445, 2015.
34. Stott, C. The Management of a 36 Year-old Man with a Frozen Abdomen Enterocutaneous Fistula and Ileostomy. Journal of Stomal Therapy Australia 38(2):8-11, 2018.
35. Song, X., Song, Y., Yuan, Y., et al. Prognostic Value of Presepsin for Outcomes and Complications in Enterocutaneous Fistula Complicated by Abdominal Sepsis. International Journal of Surgery 33:96-101, 2016.
36. Colwell, J.C. Selection of a Pouching System. In Carmel, J.E., Colwell, J.C., Goldberg, M.T., eds. WOCN® Core Curriculum: Ostomy Management. Philadelphia, PA: Wolters-Kluwer, 2016:120-30.
37. Netsch, D.S., Nix, D.P., Haugen, V. Negative Pressure Wound Therapy. In Bryant, R.A., Nix, D.P., eds. Acute and Chronic Wounds: Current Management Concepts, 5th ed. St. Louis, MO: Elsevier, 2016:350-60.
38. Limengka, Y., Jeo, W.S. Spontaneous Closure of Multiple Enterocutaneous Fistula due to Abdominal Tuberculosis Using Negative Pressure Wound Therapy: A Case Report. Journal of Surgical Case Reports 2018(1):rjy001, 2018. doi: 10.1093/jscr/rjy001.

22

Atypical Wounds

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Objectives

After completing this chapter, you'll be able to:

- recognize the importance of identifying atypical wounds
- identify clinical scenarios where an atypical wound should be suspected
- describe the various clinical manifestations of atypical wounds
- explain the need for wound biopsies in determining the etiology of an atypical wound.

Types of Atypical Wounds

The vast majority of chronic wounds are due to venous insufficiency (venous leg ulcers), long-standing diabetes mellitus (diabetic foot ulcers), prolonged pressure (pressure injury), and poor vascular supply (arterial ulcers). However, wounds resulting from other less common etiologies are referred to as *atypical wounds.*[1] Atypical ulcers are generally understood to be wounds that cannot be defined under one of the primary nonhealing wound categories.[2] These include inflammatory processes, infections, traumatic causes, metabolic disorders, malignancy, genetic diseases, and factitious injuries.[2,3]

It is of the utmost importance for the clinician to recognize when a wound is the result of an uncommon etiology since management and ultimate treatment will depend on the underlying condition.[3] A wound should be evaluated for an atypical etiology if:

- it is present in a location different from that of a common chronic wound
- its appearance varies from that of a common chronic wound
- it does not respond to conventional therapy.

As an example, the thigh is an atypical location for a pressure injury, venous leg ulcer, arterial ulcer, or diabetic foot ulcer, and therefore, the location should raise the suspicion of an atypical cause of that ulcer. Despite being a common location for venous leg ulcers, a wound on the medial aspect of the leg and extending deep into the tendon is atypical because the depth of the wound is not usual for a leg ulcer of this etiology. Finally, any wound that remains nonhealing after 3 to 6 months of appropriate treatment should raise concern for an atypical cause, even if the distribution and clinical appearance are classic for a common chronic wound.

Once a wound is deemed as atypical, the patient's history should be reevaluated for any further clues as to the underlying etiology. Even more importantly, a tissue sample (biopsy) is critical for histologic evaluation with special stains, tissue culture (for infectious

causes), and immunofluorescence testing (for some inflammatory or immune-based causes).

Evidence-Based Practice

Tissue samples are mandatory for atypical wounds because many of the unusual causes of wounds can resemble each other, making visual diagnosis alone difficult and risky.

Etiologies of Atypical Wounds

Some of the most commonly encountered etiologies for an atypical wound include inflammatory causes, infections, vasculopathies, medication induced, substance abuse related, primary skin disorders, metabolic and genetic factors, malignancies, and external causes (Table 22-1). However, a thorough medical history, including epidemiological exposure, family history, personal habits, medications, and concomitant systemic diseases, along with a thorough physical examination, histologic evaluation, and laboratory testing, will provide the critical information necessary for a correct diagnosis of an atypical wound and subsequent management.

Table 22-1 Potential Etiologies of Atypical Wounds

Although not all-inclusive, this list presents some of the most commonly encountered etiologies for an atypical wound.

Inflammatory Causes
- Vasculitis
- Pyoderma gangrenosum

Infections
- Atypical mycobacteria
- Deep fungal infections

Vasculopathies
- Cryoglobulinemia
- Cryofibrinogenemia
- Antiphospholipid antibody syndrome

Metabolic and Genetic Causes
- Calciphylaxis
- Sickle cell anemia

Malignancies
- Squamous cell carcinoma
- Basal cell carcinoma
- Lymphoma
- Kaposi's sarcoma

External Causes
- Burns
- Bites
- Stings
- Radiation
- Substance abuse related
- Medication induced

Inflammatory Causes

Among the most interesting—and probably more common—causes of atypical wounds are the inflammatory ulcers. Although a variety of inflammatory and immunologic diseases affect the skin, two relatively more common causes of inflammatory ulcers are vasculitis and pyoderma gangrenosum (PG).

Vasculitis

Vasculitis is defined as inflammation and necrosis of the blood vessels, which can ultimately result in end-organ damage—in this case, the skin.[4] Although about half of the cases are idiopathic,[5] vasculitis is a reaction pattern that may be triggered by certain reactants, among which are underlying infections, malignancy, medications, and connective tissue diseases (Table 22-2). Clinically, vasculitis varies depending on the size of the underlying vessel affected. For example, lesions may include reticulated erythema due to disease of the superficial cutaneous plexus, or they may present as widespread palpable purpura, necrosis, and ulceration due to disease in larger, deeper vessels (Fig. 22-1). Small vessel or in some cases medium-sized vasculitis such as in granulomatosis with polyangiitis or immune complex vasculitis can present with mild symptoms such as palpable purpura or more severe symptoms such as chronic ulceration. Medium-sized vessel vasculitis presents with painful nodules that often ulcerate, such as in polyarteritis nodosa. Rarely, large vessel vasculitis causes skin manifestations; however, ulceration has been reported in Takayasu arteritis. Often, vasculitic lesions on the skin are just one component of the patient's pathology—they may also have involvement of different end organs such as the kidney, lung, central nervous system, and gastrointestinal tract.[6] As such, when vasculitis is suspected, a thorough workup is indicated.

Table 22-2 Potential Etiologies of Vasculitis

Although not all-inclusive, the most common causes of vasculitis are listed below.

Infections

- *Streptococcus* spp.
- *Mycobacterium tuberculosis*
- *Staphylococcus* spp.
- *Mycobacterium leprae*
- Hepatitis viruses A, B, and C
- Herpes virus
- Influenza virus
- *Candida* spp.
- *Plasmodium* spp.
- Schistosomiasis
- HIV

Medications

- Antibiotics (penicillins, cephalosporins, minocycline, sulfa drugs)
- Granulocyte macrophage colony stimulating factor
- Propylthiouracil
- Levamisole (often found in impure cocaine)
- Nonsteroidal anti-inflammatory drugs
- Thiazides
- Allopurinol
- Tumor necrosis factor-α inhibitors

Chemicals

- Insecticides
- Petroleum products

Foods

- Milk
- Gluten

Connective Tissue and Other Inflammatory Diseases

- Systemic lupus erythematosus
- Dermatomyositis
- Sjögren's syndrome
- Rheumatoid arthritis
- Behçet's syndrome
- Cryoglobulinemia
- Scleroderma
- Primary biliary cirrhosis
- Takayasu arteritis
- Polyarteritis nodosa
- ANCA-associated vasculitis
  - Microscopic polyangiitis
  - Granulomatosis with polyangiitis
  - Eosinophilic granulomatosis with polyangiitis

Malignancies

- Lymphomas
- Leukemias
- Multiple myeloma

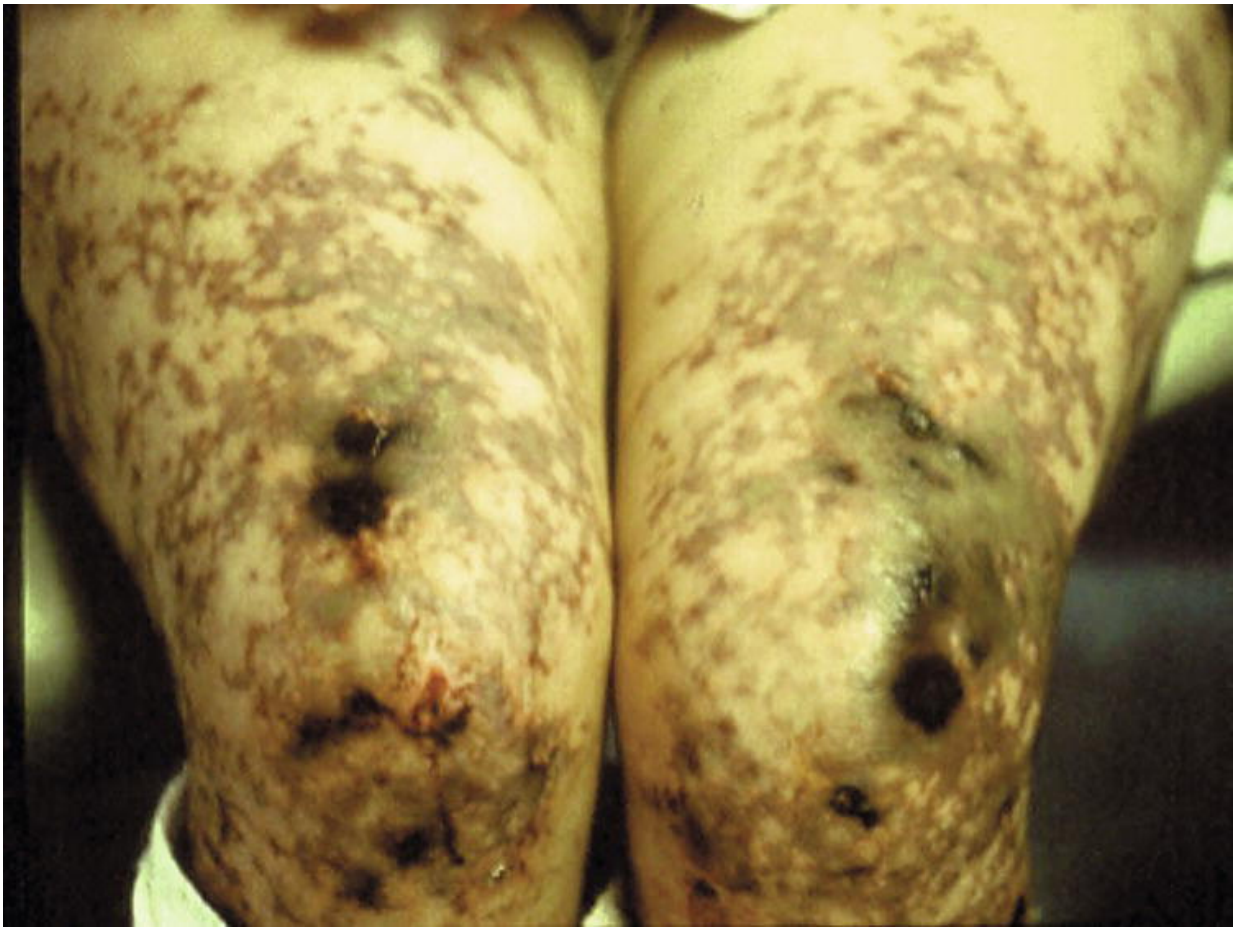

Figure 22-1. Vasculitis. This photograph shows reticulated erythema and necrotic ulcers on the thighs of a patient with vasculitis.

Circulating immune (antibody–antigen) complexes, which can deposit in the walls of blood vessels, are the cause of many types of vasculitis.[7] Tissue biopsies will confirm the presence of vasculitis if performed early, and biopsies of perilesional skin may detect the type of immunoglobulin involved in the process. However, biopsies performed later in the course of lesion development may fail to reveal immune reactants or inflammatory cells, and their by-products will degrade the immunoglobulins. Tissue culture may be helpful if the vasculitis is due to an infectious process. A histologically confirmed diagnosis mandates evaluation of other organ systems and an attempt to determine the underlying etiologic factor.

If identified—and if possible—the causative agent should be addressed. For example, a patient should be treated with antibiotics if the vasculitis is incited by a superficial infection. Additionally, treatment of the vasculitis is based on the extent of the disease (Table 22-3). Mild disease that is limited to the skin can be treated with supportive care, such as leg elevation and dressings. Treatments with limited adverse effects, such as colchicine, dapsone, antihistamines, or nonsteroidal anti-inflammatory agents, also may be used. If skin disease is extensive or systemic involvement is present, more aggressive treatment such as systemic steroids, anti-inflammatory agents, or immunosuppressants may be needed[8] (Table 22-4).

Table 22-3 Diagnostic Tests for Vasculitis

Use the following tests to determine the etiology of vasculitis.

- Antineutrophilic cytoplasmic antibody
- Rheumatoid factor
- Antinuclear antibody
- Hepatitis A, B, C profile
- Complete blood count
- Serum complement
- Antistreptolysin O titer
- Cryoglobulin level
- Serum protein electrophoresis
- Chest radiograph
- Purified protein derivative test
- Throat culture
- Tissue culture

To determine the extent of disease, use these tests:

- Urinalysis
- Stool guaiac
- Chest radiograph
- Renal function tests
- Liver function tests
- Complete blood count
- Vascular imaging

Table 22-4 Vasculitis Treatment Options

Extent of Disease	Treatment Options
Mild	• Leg elevation • Compression dressings • Antihistamines • Nonsteroidal anti-inflammatory drugs • Anti-inflammatory antibiotics • Topical steroids • Support stockings • Dapsone • Colchicine • Potassium iodide
Extensive or Systemic	• Dapsone • Systemic steroids • Stanozolol • Cyclophosphamide • Methotrexate • Azathioprine • Cyclosporine • Plasmapheresis • Mycophenolate mofetil • Tacrolimus • Other anti-inflammatory or immunosuppressant drugs

Pyoderma Gangrenosum

The term *pyoderma gangrenosum* (PG) actually refers to a disorder that is neither infectious nor gangrenous. Rather, it is an inflammatory process causing painful skin ulcers of which the etiology is not fully understood. However, it is thought to be due to a combination of neutrophilic dysfunction, genetics, and inflammation. Specifically, it is due to dysregulation of the innate immune system leading to a hypersensitivity and alterations in leukocyte chemotaxis. PG is characterized by the appearance of one or more chronic ulcerations with violaceous and undermined borders[9] (Fig. 22-2A and B). It mainly affects adults, and its usual course is that of recurring, destructive ulcers, which begin as pustules and resolve with cribriform scars. Several clinical variants of PG have been described, including ulcerative, pustular, bullous, vegetative, postsurgical (mostly following cardiothoracic and breast surgeries), medication-induced, and peristomal types.[10]

A

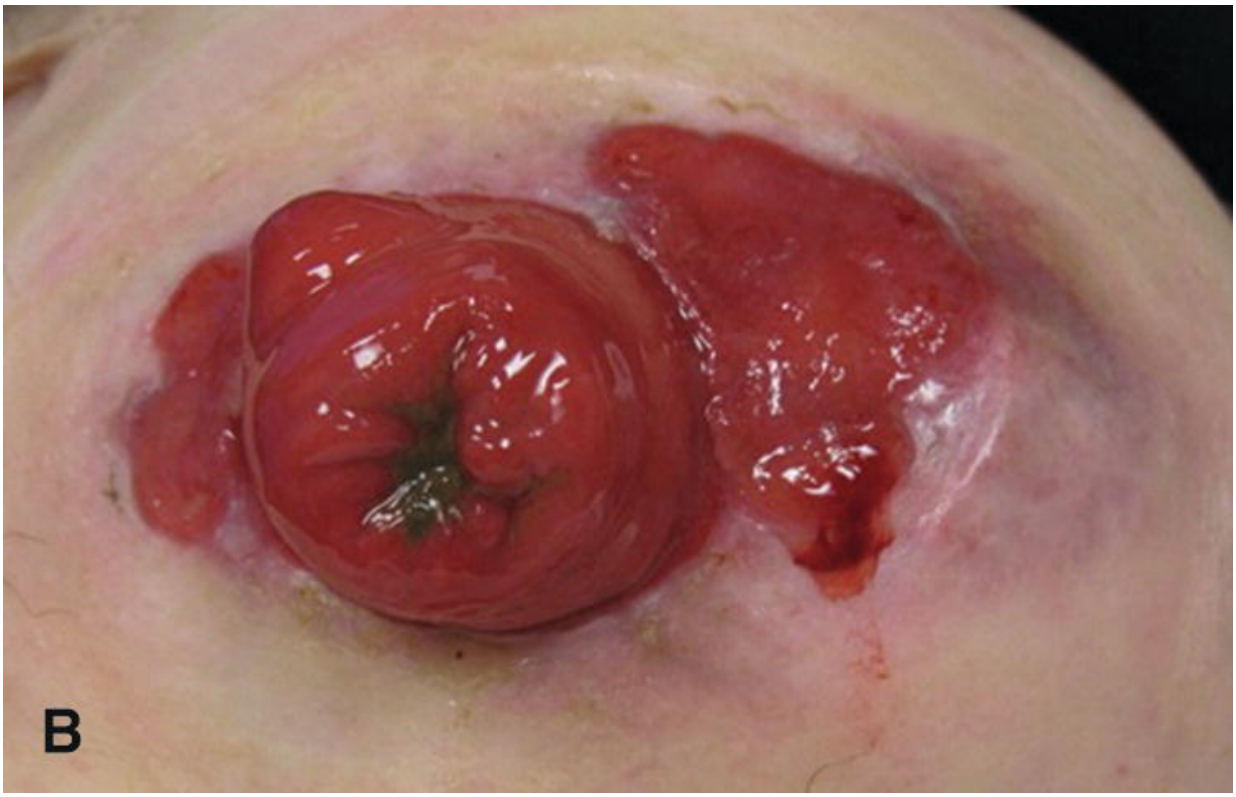

Figure 22-2. (A) Pyoderma gangrenosum in a patient with inflammatory bowel disease and pyoderma gangrenosum, this ulcer on the lateral leg shows areas of cribriform scarring. **(B)** Peristomal pyoderma gangrenosum.

Practice Point

Because a diagnostic test to confirm PG does not exist and a number of other conditions may resemble it clinically, a correct diagnosis relies on the clinical presentation and exclusion of other causes. There have been scoring systems developed such as the PARACELSUS score[11] to aid the clinician in diagnosing suspected PG.

It is important for the clinician to search for underlying diseases when a diagnosis of PG is rendered because it is associated with other conditions in more than 50% of patients[12] (Table 22-5). Among these are inflammatory bowel disease, malignancies, arthritis (seropositive and seronegative), monoclonal gammopathies, and other hematologic disorders. PG is also part of a spectrum of clinical manifestations in dermatologic syndromes such as PAPA (pyogenic arthritis, PG, and acne). PG lesions can also occur around the stoma in persons with inflammatory bowel disease[13] (Fig. 22-2).

Table 22-5 Systemic Diseases Associated with Pyoderma Gangrenosum

No diagnostic test exists to confirm the presence of pyoderma gangrenosum. In addition, pyoderma gangrenosum is typically associated with other conditions.[8] Reported associations are listed below.

Inflammatory Bowel Disease

- Ulcerative colitis
- Regional enteritis
- Crohn's disease

Arthritis

- Seronegative arthritis
- Rheumatoid arthritis
- Osteoarthritis
- Psoriatic arthritis

Hematologic Abnormalities

- Myeloid leukemia
- Hairy cell leukemia
- Myelofibrosis
- Myeloid metaplasia
- Immunoglobulin A monoclonal gammopathy
- Polycythemia rubra, polycythemia vera
- Paroxysmal nocturnal hemoglobinuria
- Myeloma
- Lymphoma

Immunologic Abnormalities

- Systemic lupus erythematosus
- Complement deficiency
- Hypogammaglobulinemia
- Hyperimmunoglobulin E syndrome
- Acquired immunodeficiency syndrome

Solid Malignancies

Pathergy (the development of ulcerations at the site of minor trauma) plays a role in the development of PG wounds. It is reported to occur in up to 40% of all cases of PG.[14] In susceptible people, even minimal trauma to the skin can result in PG lesions, such as pustules or ulcers. Caution must be taken when considering débridement for such wounds due to the risk of pathergy. As a result, gentle débridement should only be considered when the patient is adequately treated and immunosuppressed.

Curative treatment does not exist. In some patients, the course of PG waxes and wanes; however, corticosteroids usually are helpful.[15] For limited or mild disease, topical or intralesional steroids may be used. For more severe or widespread disease, systemic steroids can be used, although their adverse effects limit long-term use. In addition, immunosuppressant or anti-inflammatory agents may be of value; for example, cyclosporine appears quite effective in treating this disorder (Table 22-6). Infliximab, a monoclonal antibody to tumor necrosis factor-α, has been shown to be useful for patients with PG in a controlled, randomized, double-blinded study.[16] Infliximab is FDA approved to treat Crohn's disease, rheumatoid arthritis, ankylosing spondylitis, psoriatic arthritis, and ulcerative colitis. Additionally, topical tacrolimus and topical dapsone have been shown to be effective.[17,18] There is an increasing role for other biologic drugs and IVIG; however, studies assessing their effectiveness have been limited.

Table 22-6 Pyoderma Gangrenosum Treatment Options

Type of Treatment Options	
Topical	• Topical steroids • Topical tacrolimus • Topical dapsone • Topical cyclosporine • Nicotine patch (case reports) • Intralesional steroids
Systemic	• Steroids • Antibiotics (dapsone and minocycline) • Cyclosporine • Infliximab and other biologics • Azathioprine • Methotrexate • Chlorambucil • Cyclophosphamide • Thalidomide • Tacrolimus • Mycophenolate mofetil • Granulocyte apheresis • Intravenous immunoglobulin • Clofazimine • IVIG

Hidradenitis Suppurativa

Hidradenitis suppurativa (HS) is a chronic inflammatory condition. The exact mechanism behind the pathogenesis of HS remains unknown; however, it is thought to be due to the occlusion of the folliculopilosebaceous units.[19] As a result, HS typically affects the intertriginous areas such as the axillae, thigh, perianal, perineal, mammary, buttocks, and genital areas. HS causes the formation of inflammatory nodules, comedones, and draining sinus tracts that can result in wounds and scarring. The course of HS is relapsing and remitting, and HS causes significant morbidity to the patient due to pain, scarring, discharge, and social isolation.[20]

Diagnosis of HS is made primarily by history and the physical exam. A skin biopsy can be useful to aid in the diagnosis of HS and rule out other causes, but it is not necessary. Staging systems have been developed to categorize a patient's disease based on severity.[21] Since HS is a chronic condition, treatment is aimed at reducing new lesions, treating current lesions, and reducing scar formation. It is also imperative to decrease the patient's pain and drainage. Lifestyle changes should be made by the patient with the help of the clinician that includes weight loss, cessation of smoking, use of topical antiseptics, minimizing skin trauma, and the use of proper highly absorbent dressings to minimize the impact of the drainage. Many local and systemic drugs have been used and are being developed that are aimed at reducing inflammation and reducing bacterial load on the skin (Table 22-7). Hormonal therapy has also been used as well as surgery for extensive cases to remove the active diseased areas.

Table 22-7 Hidradenitis Suppurativa Treatment Options

- Topical clindamycin
- Intralesional steroids
- Systemic antibiotics
  - Tetracyclines
  - Clindamycin
  - Rifampin
  - Dapsone
- Hormonal therapy
- Biologic therapy
  - Adalimumab
  - Infliximab
  - Etanercept
- Apremilast
- Surgery

Infectious Causes

Infectious causes of atypical wounds may be due to a variety of different organisms, some of which are not commonly encountered in the United States. For example, atypical mycobacterial infections (other than leprosy and tuberculosis) and fungal infections (other than dermatophytes and *Candida*) occasionally are detected upon diagnostic testing. Infection caused by *Vibrio vulnificus* may be responsible for lower leg ulcers in geographic areas where there is warm salt water.

Atypical Mycobacterial Infections

Atypical mycobacteria are ubiquitous in the environment and were not generally viewed as human pathogens until the 1950s, when several cases of disease caused by these organisms were reported.[22] Cutaneous infection usually results from exogenous inoculation and predisposing factors including a history of preceding trauma, immunosuppression, or chronic disease. While *Mycobacteria marinum* is the most common agent of skin infection by atypical mycobacteria,[23] many others have been reported in recent decades (Table 22-8). The cutaneous lesions vary depending on the causative agent and may present as granulomas, small superficial ulcers, sinus tracts, or large ulcerated lesions localized in exposed areas (Fig. 22-3).

Table 22-8 *Mycobacterium* Species That Cause Skin Ulcers

Mycobacterium Species	Clinical Manifestations	Diagnosis	Treatment
M. marinum	• Swimming pool granuloma	Tissue culture	• Antituberculous drugs
M. ulcerans	• Subcutaneous nodule • Deep ulcers	Tissue culture	• Rifampin and streptomycin/ clarithromycin
M. scrofulaceum	• Cervical lymphadenitis • Fistulae	Tissue culture	• Surgical excision
M. avium-intracellulare	• Small ulcers with erythematous borders	Tissue culture	• Surgical excision • Chemotherapy
M. kansasii	• Crusted ulcerations	Tissue culture	• Antituberculous drugs • Minocycline
M. chelonae	• Painful nodules and abscesses • Surgical wound infection	Tissue culture	• Erythromycin • Tobramycin • Amikacin • Doxycycline • Clarithromycin
M. fortuitum	• Painful nodules and abscesses • Surgical wound infection	Tissue culture	• Amikacin • Doxycycline • Ciprofloxacin • Sulfamethoxazole
M. abscessus	• Abscesses • Surgical wound infection	Tissue culture	• Trimethoprim–sulfamethoxazole • Doxycycline • Levofloxacin • Clarithromycin
M. simiae	• Ulcers • Abscesses	Tissue culture	• Azithromycin • Moxifloxacin • Trimethoprim–sulfamethoxazole
M. szulgai	• Ulcers	Tissue culture	• Azithromycin • Rifampin • Ethambutol
M. xenopi	• Abscess	Tissue culture	• Azithromycin • Rifampin • Ethambutol
M. malmoense	• Rash developing into crusted lesions	Tissue culture	• Azithromycin • Rifampin • Ethambutol
M. terrae	• Abscess	Tissue culture	• Azithromycin • Ethambutol
M. haemophilum	• Skin nodularity • Necrosis	Tissue culture	• Clarithromycin • Rifampin • Rifabutin • Ciprofloxacin
M. peregrinum	• Skin nodularity • Surgical site infection • Ulcers	Tissue culture	• Clarithromycin • Levofloxacin • Ethambutol

Figure 22-3. Hansen's disease. Shown here are the leg and foot of a patient with Hansen's disease caused by *Mycobacterium leprae.* In addition to neuropathic changes of the toes and plantar aspect of the foot, this patient has a large lateral leg ulcer.

Histologically, mycobacterial infections present as granulomas and abscesses that are difficult to distinguish from those of leprosy and cutaneous tuberculosis. Diagnosis invariably will depend on tissue

culture or more recent techniques, such as polymerase chain reaction and gene rearrangement studies.

The appropriate therapy will depend on the causative agent because susceptibility to antibiotics varies and limited evidence presents a challenge to the correct treatment regimen. In some cases, simple excision of the cutaneous lesions or a combination of excision and systemic treatment often is most beneficial to the patient.

Buruli Ulcer

Buruli ulcer is a health problem in the tropical areas of many developing countries. Since 1980, Buruli ulcer has emerged as an important cause of human morbidity. It was first encountered in 1897 when Sir Albert Cook described large ulcers in patients in Uganda. In the late 1940s, MacCallum identified an organism similar to *Mycobacterium ulcerans* in an ulcer in a 15-year-old in Bairnsdale, Australia. Large ulcers were subsequently observed in patients living along the Nile River in Buruli County, Uganda, in the 1960s, hence the name of the disease. Buruli ulcer is caused by *M. ulcerans*, from the family of bacteria that causes tuberculosis and leprosy.

The World Health Organization (WHO) has defined Buruli ulcer as an infectious disease of the skin and subcutaneous tissue characterized by painless nodules, papules, plaque, or edema evolving into a painless ulcer with undermined edges and edema. Progression of the disease is associated with extensive sloughing and massive ulceration, particularly over joints, which may lead to contractures. Significant areas of the torso, face, or an entire limb may be involved. Amputation is often required when a limb is involved.[24]

Extensive cutaneous ulcers occur as the disease progresses from the early, more treatable, nodular form to the ulcerative form, which can result in the loss of skin and soft tissue. Although Buruli ulcer is rarely fatal, the associated disabilities and disfigurement are extensive and have a profound impact on quality of life in affected patients[24,25] (Fig. 22-4).

Figure 22-4. Buruli ulcer. This photo shows extensive sloughing and massive ulceration, typically leading to contractures and extensive disability and disfigurement.

The majority of those suffering from Buruli ulcer are between 5 and 15 years of age, but it can affect patients of any age. A prospective study showed that 8-week regimen of rifampicin and streptomycin (injectable) for patients with all stages of Buruli ulcer led to healing in 93% of patients without the need for surgery.[26] A more recent prospective study determined that clarithromycin (oral) could replace the injectable streptomycin after 2 weeks of standard rifampin/streptomycin treatment without loss of efficacy.[27] The WHO recently developed a categorization system to assist in the management and prognosis of Buruli ulcers that is largely based on the size of the ulcerations.[28] Unfortunately, many patients need

surgical débridement and skin grafting even when systemic antibiotics are used.

Healing often is slow and can be associated with significant functional incapacity due to contractures and amputation. Patients must be followed closely as the lesions may not heal. The course of disease can be complicated by the ability of the organism to suppress the immune system of the host. Disease progression is related directly to a decrease in the level of interferon-γ, a Th1 cytokine, as well as a concomitant increase in the Th2 cytokine interleukin-10.[24] Reversing the deficiency of interferon-γ and/or decreasing the interleukin-10 level has been identified as a novel therapeutic target for interventional development.

The disease, its treatment, and the resulting disabilities are a significant burden to patients, their families, and their society. In some countries such as Ghana, patients and families are reluctant to seek treatment due to lack of money or misunderstanding that treatment can lead to amputation.[25] Educational sessions for clinicians to recognize Buruli ulcer early and to change patient, family, and societal resistance to seeking early treatment have met with success, and the number of late cases of this disease in Ghana has declined over the past decade.[24,25]

Deep Fungal Infections

Deep fungal infections of the skin can be divided into subcutaneous and systemic mycoses. The subcutaneous mycoses result from traumatic implantation of the etiologic agent into the subcutaneous tissue, development of localized disease, and eventual lymphatic spread. In rare instances, hematogenous dissemination can occur, especially in immunocompromised hosts. As may occur with sporotrichosis or chromomycosis, ulcers from deep fungal infections are found worldwide and can present in a wide variety of clinical settings.[29]

Systemic mycoses are the result of systemic penetration of pathogenic fungi, with the lungs being the most common port of entry. These infections are restricted to the geographic areas where the fungi occur, especially tropical regions such as Central and South

America. After an initial pulmonary infection, the fungi can spread hematogenously or via lymphatic vessels to other organs, including the skin. A decrease in immunity will lead to expression of the fungal infection, as is commonly observed in patients infected with human immunodeficiency virus (HIV).

Practice Point

A thorough patient history assists the clinician in considering a diagnosis of systemic mycosis because of the limited area in which the causative fungi occur.

Sporotrichosis

Sporotrichosis is a subacute or chronic fungal infection caused by the fungus *Sporothrix schenckii.* Occurring as a consequence of traumatic implantation of the fungus into the skin, it is often associated with lymphangitis. Less commonly, inhalation of the conidia can lead to pulmonary infection and subsequent spread to the bones, eyes, central nervous system, and viscera. Systemic disease is seen in individuals with impaired immunity, such as alcoholics and AIDS patients.[30]

S. schenckii, a saprophyte in the environment, has been isolated in a variety of plants and other fauna as well as in animals (bites or scratches from animals, such as armadillos and cats). Individuals whose professional or leisure activities (such as rose gardening or landscaping) expose them to the environment are at greater risk of acquiring the infection. Lymphocutaneous sporotrichosis begins as a papule, which then ulcerates with time. The lesions spread and occur via lymphatic channels traveling from the inoculation site. Sporotrichosis is treated with systemic medications, including saturated solution of potassium iodide, itraconazole, terbinafine, and

amphotericin B. Topically applied heat also may be used because the organism grows at low temperatures.

Chromoblastomycosis

Chromoblastomycosis is a subcutaneous mycosis caused by several pigmented fungi, including *Fonsecaea pedrosoi*, *Fonsecaea compacta*, *Phialophora verrucosa*, *Cladosporium carrionii*, and *Rhinocladiella aquaspersa.* These fungi are acquired through inoculation of the causative agents in the skin, after which a mycotic infection develops at the site of entry. These microorganisms can be found in soil throughout the world; however, the disease is most common in tropical and subtropical climates, with the majority of cases seen in South America.[31]

Primarily affecting men ages 30 to 50, the principal lesion is a slow-growing papule that eventuates into a verrucous nodule. Exposed areas are involved, with extremities—especially the lower limbs—being affected in 95% of the cases.[29] The surface of the lesion may be covered by scales or may be ulcerated with serosanguinous crusts. Black dots can be often observed; these dots are rich in fungi and represent the site of transepidermal elimination of necrotic tissue.

Diagnostic examinations should include scrapings from the lesion with potassium hydroxide 20%; tissue samples should be obtained from biopsies for tissue culture and histology.

The disease tends to be chronic and difficult to treat and may lead to lymphedema and elephantiasis. Ulcerated and cicatricial lesions have been reported to develop into carcinoma. Small lesions can be cured by surgical excision; however, chronic lesions are often resistant to treatment.

Systemic antifungal agents, such as ketoconazole, itraconazole, terbinafine, and amphotericin B, have been used, both alone and in combination, with variable results. Oral flucytosine, alone or in combination with an antifungal, has also been reported to be therapeutic.[31] The new-generation triazole antifungals show promise, as demonstrated by the positive results with posaconazole, although they may be prohibitively expensive.[32,33] Cryosurgery has also been

used alone and in combination with antifungal chemotherapy. In addition, local heat therapy of 42°C to 45°C can also be an effective therapeutic modality.

Paracoccidioidomycosis

Paracoccidioidomycosis (South American blastomycosis) is a chronic, infectious disease caused by the fungus *Paracoccidioides brasiliensis*, a saprophyte of soil and decaying vegetation found in tropical and subtropical climates. Infection occurs primarily via a respiratory route with occasional dissemination to other organs, including the skin. Rarely, direct inoculation into the skin can occur. Patients present with painful ulcerative lesions of the mouth, the face, or, less frequently, the extremities. Involvement of regional lymphatics is characteristic.[34]

Diagnosis can be established by isolation and identification of the etiologic agent with direct mycologic examination, histopathologic or cytopathologic examination, or culture. Treatment includes trimethoprim–sulfamethoxazole, azole derivatives such as itraconazole and ketoconazole, and amphotericin B for severe cases.

Mycetoma

Mycetoma is a chronic infection of the skin and subcutaneous tissue characterized by local edema, sinus tract formation, and the presence of grains—hard concretions representing colonies of the etiologic agent. It occurs worldwide but most commonly in tropical and subtropical regions. Mycetomas can be divided into eumycetomas, caused by fungi, and actinomycetomas, caused by actinomycetes. The most common agent in Central and South America is the bacteria *Nocardia brasiliensis*, which are found in soil.[35] This agent is rarely found in the United States, but when it does occur, the fungus *Pseudallescheria boydii* is the most commonly isolated agent.

Male rural workers ages 20 to 40 are most frequently affected. After trauma, a slow-growing, painless nodule develops, which may discharge purulent material and grains. Neighboring lesions may interconnect with each other, giving rise to the sinus tracts that are characteristic of the disease.

Diagnosis can be established based on clinical findings; additional examinations may include visualization of grains or filaments in discharge or biopsy and tissue culture. On ultrasonographic evaluation, the mycetoma grains, capsules, and the resulting inflammatory granulomas have characteristic appearances.[36] Treatment is difficult. Surgical excision, commonly in combination with systemic therapy, may be effective. Sulfonamides, tetracycline, aminoglycosides, rifampin, ciprofloxacin, amoxicillin, clavulanate, and oral azoles may be used depending on sensitivities of the etiologic agent. Recent preliminary evidence suggests that linezolid, imipenem, and the newer triazoles voriconazole and posaconazole also may be efficacious.[37]

Cryptococcosis

Cryptococcosis is caused by *Cryptococcus neoformans* and *Cryptococcus gattii*. Classically, this fungus infects HIV patients who are immunocompromised with meningitis. When disseminated, it causes maculopapules, ulcers, pustules, and abscesses. However, sole cutaneous manifestations are reported in up to 18% of patients with solid organ transplants.[38] Diagnosis of primary skin infections should be obtained with a tissue culture and special staining.

When infected, these patients present with necrotizing soft tissue infections and cellulitis. These primary skin infections should be treated with fluconazole.[39]

Phaeohyphomycosis

Phaeohyphomycosis is a group of mycotic infections that include *Exophiala* species, *Alternaria* species, and *Cladophialophora* species. These can infect both immunocompromised and immunocompetent patients. Lesions consist of nodules, plaques, and ulcers and are becoming more prevalent due to the increase in solid organ transplantation. Diagnosis should be confirmed with tissue culture and special staining. First-line therapy should include itraconazole, voriconazole, or posaconazole. In many cases, surgical débridement is necessary.[40]

Vibrio vulnificus Infection

V. vulnificus, a bacterium, is found widely in raw shellfish in Atlantic Coast waters.[41] It produces a wide variety of extracellular toxins that favor tissue invasiveness. The bacteria utilize iron and their virulence is increased in patients with higher transferrin levels.[42] Wound infection with *V. vulnificus* occurs when contaminated seawater enters the body through a break in the epidermal barrier, commonly during fishing or water sport activities. Breaks in the skin relating to opening oysters are a common method of inoculation. Pustular lesions, lymphangitis, lymphadenitis, and cellulitis may ensue; in some cases, rapid progression to myositis and skin necrosis (necrotizing fasciitis [NF]) follows. Diagnosis is made with wound cultures. Treatment of *V. vulnificus* wound infections consists of antibiotics, such as the combination of minocycline or doxycycline and cefotaxime or ceftriaxone, and wound care. Aggressive débridement may be necessary. For more minor infections, tetracycline or fluoroquinolone monotherapy may be used.[43]

Primary septicemia from *V. vulnificus* occurs 24 to 48 hours after the ingestion of raw or undercooked seafood, especially raw oysters. Patients with liver disease, hereditary hemochromatosis, and chronic diseases such as renal failure or diabetes mellitus are especially at risk for infection.[44] Clinically, fever and hypotension may be present, along with the development of bullous cellulitis and necrotic skin ulcers.

Necrotizing Fasciitis

NF is an uncommon but life-threatening soft tissue infection characterized by rapidly spreading inflammation and necrosis of the skin, subcutaneous fat, and fascia[45] (Fig. 22-5). Mortality rates have exceeded 70% in the past,[46] although recent data have shown a dramatic decrease, with mortality rates below 10%.[47] Rapid, early intervention, including wide surgical débridement and antimicrobial therapy, is imperative to reduce morbidity and mortality.

Figure 22-5. (A) Necrotizing fasciitis of the foot. **(B)** Necrotizing fasciitis of the abdomen.

The incidence of NF has been reported to be 0.40 cases per 100,000 population.[47] Although it is rare, certain conditions can predispose patients to developing the disease, including diabetes mellitus (the most common), acquired immunodeficiency syndrome (AIDS), malignancy, obesity, peripheral vascular disease,[48] and trauma, such as burns, lacerations, or minor trauma (Table 22-9).

Table 22-9 Conditions Leading to Necrotizing Fasciitis

The conditions listed below place patients at risk for NF[29,33,34]

- Age 50 and older
- Alcoholism
- Anogenital infection: anorectal, perianal, scrotal, ischiorectal, periurethral abscess
- Atherosclerosis: coronary artery disease, peripheral vascular disease
- Chronic obstructive pulmonary disease
- Cirrhosis or chronic liver disease
- Compromised skin integrity: psoriasis, herpes zoster, chronic leg ulcers
- Diabetes mellitus
- Hypertension
- Immunosuppression: HIV, corticosteroid therapy
- Intravenous drug use
- Malignancy
- Malnutrition
- Obesity
- Renal failure
- Smoking
- Surgery
- Trauma: needle puncture, insect bites, fish-fin injury, burns, lacerations, surgical wounds

NF spreads quickly along the fascial planes due to the relatively poor blood supply to this area. Thus, initially, overlying tissue may appear healthy even though the infection is rapidly spreading. NF is

categorized as type 1, type 2, or type 3 depending on which organisms are cultured. Type 1 NF is caused by a polymicrobial infection from aerobic and anaerobic bacteria such as *Clostridium* and *Bacteroides* species. Type 2 NF consists of group A *Streptococcus* (*Streptococcus pyogenes*) with or without a coexisting staphylococcal infection. Type 3 NF is caused by a *Vibrio* infection from a puncture wound due to fish or marine insects.

NF can affect any area of the body, but it most commonly occurs on the extremities. Involvement of the genitalia is referred to as Fournier's gangrene (a urological emergency) and usually results from a polymicrobial infection.[49] There is a higher mortality rate when the head, neck, chest, and abdomen are involved because those areas tend to be refractory and, as such, are more difficult to treat.

Early on, patients generally present with a clinical picture similar to cellulitis. As with less aggressive cases of cellulitis, redness and edema can be seen at the site, with a spreading, diffuse inflammatory reaction that blends into the surrounding tissue. The overlying skin is shiny and tense without any clear lines of demarcation. Crepitus is often present due to bacterial gas formation below the skin. Patients complaint of severe pain are usually out of proportion to the physical exam. This characteristic clue of NF may be the only hint of a deeper, more aggressive infection. It is critical to be alert for this sign because the earlier the diagnosis is made, the earlier treatment can be instituted and the better the chance of survival will be. Radiological imaging can assist in diagnosis; however, when NF is suspected, patients should go immediately to the operating room for surgical exploration. Treatment involves immediate surgical intervention to remove infected and necrotic tissue and mass irrigation in addition to systemic antibiotic therapy.[50]

Over time, frank cutaneous gangrene may extend beyond the skin and into the subcutaneous fat and fascial planes below. Separation of the necrotic tissue along the fascial planes with suppuration may occur. Myonecrosis develops in the underlying muscle. Lymphadenitis, lymphangitis, crepitation, and venous thrombosis are seen less often.

Metastatic abscesses have been reported in the liver, lung, spleen, brain, and pericardium, but these are rare. In addition to cutaneous manifestations of NF, there are systemic findings as well as those associated with progression of disease. Patients usually will appear toxic with high fever, chills, and constitutional symptoms. In fulminant cases, multiorgan system failure occurs.

Vasculopathies

A heterogeneous group of disorders is classified under this category. Vasculopathy is characterized by microvascular occlusion of vessels within the skin due to thrombi or emboli, which leads to tissue hypoxia and the clinical manifestations of purpura, livedo reticularis (mottled reticulated vascular pattern), and painful ulcers. Cryofibrinogenemia, monoclonal cryoglobulinemia, cholesterol emboli, and antiphospholipid antibody syndrome (APS) are among the causes of vasculopathies that commonly present as atypical skin ulcerations of the lower extremities (Fig. 22-6).

Figure 22-6. Vasculopathy.

Cryofibrinogenemia

Cryofibrinogenemia occurs as a primary (idiopathic) disorder or in association with underlying diseases, such as infectious processes, malignancy, or collagen, vascular, or thromboembolic disease. The clinical presentation is painful cutaneous ulcerations located on the leg and foot; these lesions are usually unresponsive to treatment (Fig. 22-7A and B). Other cutaneous findings include livedo reticularis, purpura, ecchymoses, and gangrene. The pathogenesis of the lesions is related to the in vivo occlusion of small blood vessels initiated in the distal extremities by the abnormal precipitate. This hypothesis is corroborated by the pathology findings of cryofibrinogen, consisting of thrombi within superficial dermal vessels due in part to protein deposition. Cryofibrinogen is a circulating complex of fibrin, fibrinogen, and fibronectin along with albumin, cold-insoluble globulin, and factor VIII. The complex is soluble at 98.6°F (37°C) but forms a cryoprecipitate at 39.2°F (4°C).[51] Additionally, this complex can be made to clot with thrombin. The mechanism by which cryofibrinogen is produced is not well understood. Diagnosis is made with tissue biopsy. Treatment is symptomatic (avoiding cold, smoking cessation) or, in secondary disease, directed to the underlying cause.[52] Agents that lyse fibrin thrombi are helpful.

A

B

Figure 22-7. (A) Cryofibrinogenemia. **(B)** This patient has painful punctate ulcers on the feet and legs secondary to cryofibrinogenemia.

Stanozolol, streptokinase, and streptodornase have been used with success.

Cryoglobulinemia

Cryoglobulinemia occurs when deposits of cryoglobulins lead to the formulation of thrombi in medium and small vessel walls.[53] Three types of cryoglobulinemia have been identified: type I or monoclonal cryoglobulinemia may be seen in patients with malignant diseases, such as multiple myeloma, or benign lymphoproliferative conditions, such as Waldenström's macroglobulinemia. This classically leads to thrombotic phenomena but can clinically resemble vasculitis. Type II or mixed cryoglobulinemia combines polyclonal and a monoclonal immunoglobulin; this type of cryoglobulinemia is seen less often in association with malignancies and more frequently with infectious or inflammatory diseases. Type III is comprised of only polyclonal immunoglobulin and is most commonly associated with hepatitis C infection. Both type II and III cryoglobulinemia cause vasculitis, which can lead to skin ulcers.[54] Other skin manifestations include acral cyanosis, Raynaud's phenomenon, livedo reticularis, altered pigmentation of involved skin, and palpable purpura, which may progress to blistering and frank ulceration. Some patients may have systemic manifestations, such as arthritis, peripheral neuropathy, and glomerulonephritis. Diagnosis is based on skin biopsies, which show either vasculopathy or vasculitis, and subsequent detection of cryoprecipitate and analysis of cryoglobulins by immunoelectrophoresis.

Practice Point

Evaluate the patient with vasculitis for type II or III cryoglobulinemia; these conditions can cause vasculopathy.

Treatment should be directed at the underlying cause in cryoglobulinemia. For example, interferon-α, ribavirin, and newer direct-acting antivirals are used to treat hepatitis C and may result in resolution of the associated cryoglobulinemia.[55] Corticosteroids alone or in combination with immunosuppressive drugs (cyclophosphamide or mycophenolate mofetil) have been used to induce remission in patients with cryoglobulinemia. Plasmapheresis and rituximab can be used as well to treat severe cases of cryoglobulinemic vasculitis.[56]

Cholesterol Crystal Embolism

Cholesterol crystal emboli occur when cholesterol plaque and crystals embolize and occlude small peripheral vasculature. These can occur spontaneously; however, an increasing amount occurs following instrumentation within the vasculature, such as angioplasty or catheterization.[57] Clinical manifestations depend on the location of the occlusion such as renal, central nervous system, or gastrointestinal vasculature. However, the most common vessels occluded cause cutaneous symptoms such as gangrene, cyanosis, purpura, petechiae, livedo reticularis, and ulceration. Diagnosis is confirmed with a biopsy. Treatment for the cutaneous manifestations is directed toward preventing recurrence with lipid-lowering therapy such as statins. Usually, occluded vessels impacting the skin cannot be surgically revascularized. Additionally, these patients should not be anticoagulated due to risk of hemorrhage.[58]

Antiphospholipid Antibody Syndrome

APS is characterized by elevated titers of different antiphospholipid antibodies in association with venous or arterial thrombosis, recurrent fetal loss, and often thrombocytopenia. Antiphospholipid antibodies include lupus anticoagulant, anticardiolipin antibodies, and anti–β_2-glycoprotein-1 antibodies. All or any of the antibodies are part of the syndrome.[59,60] APS may present as primary disease or may be secondary to an underlying autoimmune disease such as systemic lupus erythematosus (seen in about 50% of cases). Elevated antiphospholipid antibody titers also have been associated with malignancy and infectious states. The exact pathogenic mechanism of APS remains unclear, but it is thought that once the antiphospholipid antibodies form, they act directly upon coagulation pathways and increase the susceptibility to thromboses. In addition, they may increase vascular tone.[61]

The clinical hallmark of this disease is the presence of livedo reticularis. Arterial and venous thrombosis may elicit a variety of skin lesions, including ulcerations (most commonly), superficial thrombophlebitis, and cutaneous infarcts. Any organ system can be affected. Placental vessel thrombosis and ischemia can result in miscarriage precipitated by placental insufficiency. A proposed mechanism responsible for this event is a disruption of annexin A5 (a cell surface protein with anticoagulant properties) binding of phospholipids on the trophoblast cells by antiphospholipid antibodies, thus promoting a procoagulant state that leads to thrombosis at the maternal–fetal interface followed by damage to the placenta and eventual fetal loss.[62] Treatment includes the use of aspirin, warfarin, and prednisone, but response is not uniform.

Metabolic Disorders

Metabolic diseases are uncommon causes of chronic wounds. One condition, called calciphylaxis, is commonly seen in a subset of patients undergoing long-term hemodialysis who develop secondary hyperparathyroidism.[63] This leads to deposition of calcium within soft tissue and arteries and, eventually, tissue death.

Calciphylaxis

Calciphylaxis is a rare, often fatal condition that is characterized clinically by progressive cutaneous necrosis, which frequently occurs in patients with end-stage renal disease. Many eliciting factors have been suggested, but the most common linking phenomenon is the development of secondary hyperparathyroidism.[64] Secondary hyperparathyroidism causes elevated calcium–phosphate product and the development of vascular, cutaneous, and subcutaneous calcification that, in turn, leads to tissue death. Interestingly, most patients with severe hyperparathyroidism do not have skin necrosis; thus, other less well-understood mechanisms are thought to play a role in the pathogenesis of calciphylaxis. Calciphylaxis is seen in approximately 1% to 4% of patients with ESRD on dialysis.[65,66] The prognosis for patients who develop calciphylaxis is grim, with an estimated 1-year survival rate of approximately 46%.[67,68] In addition to skin involvement, the pathophysiologic process may also occur within internal organs; this, along with sepsis from infected skin wounds, is a major cause of morbidity and mortality in patients with this condition.

The cutaneous manifestations usually begin with pain and the development of red or violaceous mottled plaques in a livedo reticularis–like pattern. This signifies a vascular pattern, and these early ischemic lesions often progress to gangrenous, ill-defined, black plaques (eschars) (Fig. 22-8A). With time, the plaques ulcerate and become tender, indurated ulcers can lead to autoamputation. The ulcers of calciphylaxis are usually bilateral and symmetric and may

extend deep into the muscle. Vesicles frequently appear at the periphery of the ulcers (Fig. 22-8B).

Figure 22-8. (A) Defined, black plaques of calciphylaxis. **(B)** Calciphylaxis lesion that has ulcerated.

The term *calciphylaxis* is considered a misnomer that originated from experiments in rats that resulted in interstitial calcification.[69] Calciphylaxis should be distinguished from calcinosis cutis, which may be associated with significant morbidity but typically not mortality. Calciphylaxis has been given a number of other names, including uremic gangrene syndrome, metastatic calcinosis, azotemic calcific arteriopathy, and calcific uremic arteriopathy. Some authors prefer the term *calcific uremic arteriopathy*[70] because it reflects the histologic findings of calcium deposits in the walls of the small- and medium-

sized vessels of lesions. The important distinction is that calcific uremic arteriopathy involves small-vessel calcifications with intimal hypertrophy and small-vessel thrombosis (Fig. 22-9).

B

C

Figure 22-9. Calcific uremic arteriopathy (CUA). **(A)** CUA with livedo reticularis pattern. **(B)** CUA of the thigh. **(C)** CUA of bilateral extremities. (Courtesy of D. Beless, MD.)

The pathogenesis of calciphylaxis is still poorly understood. Recent studies have shown increases in the expression of bone-specific proteins, including collagen I, bone morphogenetic protein 2 and 3, matrix Gla protein, osteopontin, osteonectin, and osteocalcin in calcified arteries. These proteins, together with elevated calcium and inorganic phosphate levels, may induce calcification or osteoblastic differentiation in pluripotent vascular smooth muscle cells.[63,64,70,71]

Diagnosis can usually be made based on clinical findings. Although vascular processes may present similarly, laboratory evaluation for the presence of an elevated calcium, phosphate, or calcium × phosphate product confirms calciphylaxis. An elevated intact parathyroid hormone level along with radiographic evidence and consistent histology also help confirm a diagnosis. Histology shows dermal and pannicular arteriolar calcification, subintimal fibrosis, and thrombotic occlusion.

As secondary infections may have tragic sequelae, treatment of infected ulcers is critical. Swab and tissue cultures from the wound may aid in guiding antibiotic therapy.

The treatments used for patients with calciphylaxis can be divided into either medical or surgical therapies[63,64,70,71] and are often *used in tandem* (Table 22-10).

Table 22-10 Calciphylaxis Treatment Options

Medical Treatment Options

- Phosphate binders
- Decreased calcium in dialysate
- Antibiotics
- Low-phosphate diet
- Cinacalcet
- Bisphosphonates
- Sodium thiosulfate
- Avoidance of challenging agents
- Avoidance of systemic steroids
- Hyperbaric oxygen
- Anticoagulation
- Cyclosporine
- Stanozolol

Surgical Treatment Options

- Parathyroidectomy
- Wound care and débridement
- Amputation
- Renal transplantation
- Skin grafting using either autologous or tissue-engineered skin

Malignancies

Malignancies may present as either wounds or developing from wounds. Nonmelanoma skin cancer, lymphomas, and sarcomas may ulcerate as they outgrow their blood supply (Fig. 22-10). Alternatively, chronic wounds may develop into a malignancy, most commonly squamous cell carcinoma. This phenomenon is termed *Marjolin's ulcer* after the author who first described cells from an edge of a chronic wound that had undergone malignant change—an occurrence that can be seen in up to 2% of chronic wounds[72] (Fig. 22-11). A similar phenomenon may also occur in scars, burn wounds, sinus tracts, chronic osteomyelitis, and even vaccination sites.

Figure 22-10. Malignancies. Shown here is a right medial leg ulcer in a venous distribution secondary to T-cell lymphoma.

Figure 22-11. Marjolin's ulcer. Shown here is a chronic wound that developed malignant changes (squamous cell carcinoma).

The precise mechanism of malignant degeneration in chronic wounds is not known, although several theories have been proposed.[73,74] One such theory is that the weakened epithelium and the decreased vascularity of the wound make it susceptible to carcinogens. Another postulated idea is that possible toxins released from the wound tissue lead to mutations and eventual carcinogenesis. In addition to squamous cell carcinoma, basal cell carcinomas and other neoplasms, such as Kaposi's sarcoma and lymphoma, have also been found in chronic wounds.

Early identification of malignancy, typically via biopsy of suspected lesions, is critical. Treatment of biopsy-confirmed neoplasia includes excision with margins (usually at least 2 cm), possibly adjuvant therapy with radiation, topical 5-fluorouracil, methotrexate, L-phenylalanine mustard, and even lymph node dissection; amputation of the affected limb may be necessary in some cases.[73] Mohs micrographic surgery to ensure complete removal of the primary lesion has been used successfully to treat malignancy arising from chronic osteomyelitis.[75]

External Causes

External causes of atypical wounds include spider bites, chemical injury, chronic radiation exposure, trauma, and factitial ulcers. A thorough patient history is the most valuable tool in determining the etiologic agent in ulcers caused by external factors.

Spider Bites

At least 50 spider species in the United States have been implicated in causing significant medical conditions; however, the *Loxosceles* (brown recluse or violin spider) species are the most well known to cause skin necrosis and ulcers in the Americas.

Loxoscelism

The bite of *Loxosceles reclusa* is usually painless but can produce pain and a burning sensation. It often goes unnoticed; however, some patients will experience progression to more significant wounds. The venom produced by this species contains phospholipase D and sphingomyelinase; the latter is thought to be responsible for the cutaneous manifestations. In these patients, enlargement of the bite site occurs within 2 to 6 hours, with associated pain and general symptoms, such as fever, malaise, headache, and arthralgia. As the disease progresses, a pustule, blister, or large plaque forms at the bite site. These wounds may present as a deep purple plaque surrounded by a clear halo (vasoconstriction) and surrounding erythema—the so-called red, white, and blue sign (Fig. 22-12). It usually heals within weeks, but in a small percentage of cases, necrosis develops and may take months to heal.[76] Viscerocutaneous loxoscelism, a systemic illness that can include diarrhea, nausea, vomiting, petechia, urticaria, and disseminated intravascular coagulation, can occur in 0.7% to 27% of patients depending on the geographic region. These systemic findings do not correlate with the severity of the local dermatologic symptoms.[77]

Figure 22-12. Loxoscelism. Bite from a brown recluse spider (*Loxosceles reclusa*). Note the deep purple plaque surrounded by a clear halo and erythema, the so-called red, white, and blue sign.

With bites occurring in areas of greater fat content, such as the abdomen, buttocks, and thighs, necrosis develops more frequently. When the eschar is shed, an ulcer may result. Healing of the ulcer is generally very slow and may take up to 6 months.

The differential diagnosis includes foreign body reaction, infections, trauma, vasculitis, PG, and methicillin-resistant *Staphylococcus aureus* infection. Treatment consists of cooling the bite site, elevation (if possible), and analgesics. The use of dapsone and corticosteroids is controversial. In cases of necrosis, surgical débridement may be warranted.[61]

Chemical Burns

A variety of chemical products are capable of producing skin wounds.[78] Cutaneous injury caused by caustic chemicals progresses continually after the initial exposure and, if not properly cared for, may produce painful ulcers that are difficult to heal. The lesions caused by alkalis are usually more severe than those caused by acids; however, the severity of the burn is determined by the mode of action and concentration of the chemical as well as the duration of contact before treatment is initiated.[78] Prolonged irrigation with water for 30 minutes (for most chemicals) or longer is the most important initial treatment, followed by standard burn care.

Certain chemicals possess unique properties that require special additional therapy, such as hydrofluoric acid (application of 25% magnesium sulfate), chromic acid (excision of the affected area), and phenol (application of polyethylene glycol mixed with alcohol 2:1).[78]

Radiation Dermatitis

After exposure to ionizing radiation exceeding 10 Gy, local skin reactions characterized by mild erythema, edema, and pruritus may occur. This acute radiation dermatitis usually begins 2 to 7 days after exposure, peaks within 2 weeks, and gradually subsides. With exposure to higher doses, intense erythema with vesiculation, erosion, and superficial ulceration may ensue. Postinflammatory pigmentary abnormalities (usually hypopigmentation), telangiectasia, and atrophy are common.[79,80]

Hyperbaric oxygen is a possible treatment option for ulcers caused by radiation.

Factitial Dermatitis

The term *factitial dermatitis* denotes a self-imposed injury. The clinical appearance of these ulcers is usually particular, with sharp or linear edges in an area of easy access such as the extremities, abdomen, and anterior chest (Fig. 22-13).

Figure 22-13. Factitial dermatitis. This photograph shows an angulated factitial ulcer on the breast. The term *factitial* denotes a self-imposed injury.

The care includes evaluation and treatment of underlying psychological diseases and limitation of accessibility to the wound, such as placing a dressing or cast over the wound. These patients would often benefit from close follow-up from both dermatologists and psychiatrists. Additionally, these patients may benefit greatly from a clinician who specializes in psychodermatology. Often, there are medications that can alleviate the urge for self-injurious behavior.

Drug-Induced Causes

Coumadin Necrosis

Coumadin (warfarin)-induced skin necrosis is a rare complication of anticoagulant treatment. It is estimated that 1 in every 1,000 to 10,000 patients treated with this agent experiences this complication. Coumadin skin necrosis occurs almost exclusively between the 3rd and 10th day after beginning anticoagulation therapy in association with the administration of a large initial dose of the drug. Receiving the initial dose of Coumadin without concomitant heparin also increases the risk of necrosis. Incidence of Coumadin necrosis has decreased with the implementation of bridging with another anticoagulant such as heparin when initiating Coumadin. Although the precise nature of the disease is still unknown, advances in knowledge about protein C, protein S, and antithrombin III anticoagulant pathways have led to a better understanding of the mechanisms involved in pathogenesis, which is thought to be due to a transient hypercoagulable state due to Coumadin's initial impact of inhibiting antithrombotic proteins prior to the inhibition of prothrombotic proteins.[81]

Postpartum women have a unique risk due to reduced levels of free protein S during the antepartum and immediate postpartum periods.[82] Manifestations range from ecchymosis and purpura to hemorrhagic necrosis; maculopapular, vesicular urticarial eruptions; and purple toes in all affected patients.

Wounds are painful and usually evolve into full-thickness skin necrosis within a few days. Differentiating between warfarin/Coumadin-induced skin necrosis and NF, gangrene, and other causes of skin necrosis may be difficult.[81]

Cessation of warfarin administration is the first step in treatment, with heparin used instead if anticoagulation is needed. Local wound care can be conservative but may include débridement and grafting, depending on the extensiveness of the wound. Previously uncomplicated courses of warfarin therapy do not obviate the

possibility of skin necrosis with future warfarin administration. Initiation of low-dose warfarin with heparin can reduce the likelihood of this disorder.

Extravasation

Solutions of calcium, potassium, bicarbonate, hypertonic dextrose, cardiac drugs, chemotherapeutic drugs, cytotoxic drugs, and antibiotics can lead to extravasation injury. Tissue loss can evolve into extensive wounds. Local wound care with débridement and eventual skin grafting is usually required for extensive skin and tissue loss (Fig. 22-14A–C).

B

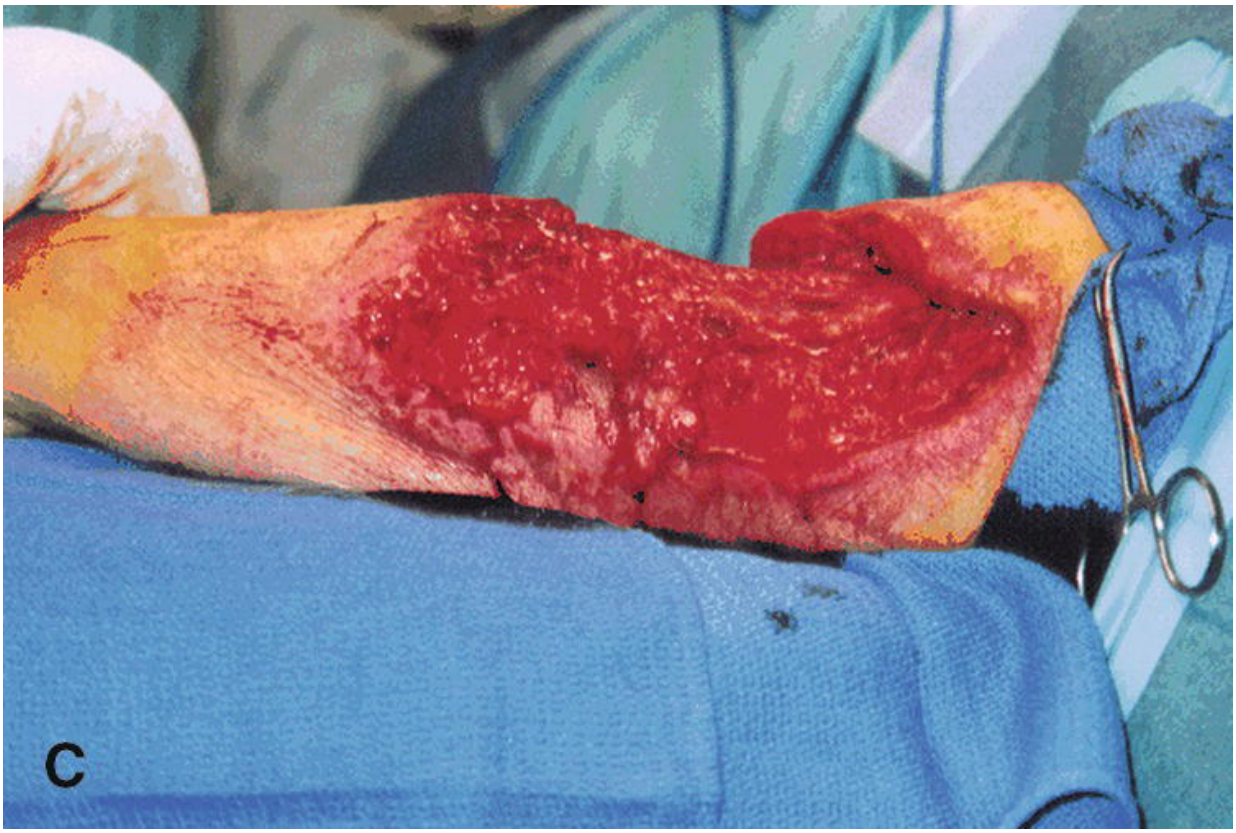
C

Figure 22-14. Extravasation. **(A)** Extravasation can cause tissue loss that may evolve into extensive wounds, as shown in this IV site 24 hours after infiltration of calcium chloride. **(B)** The same site 48 hours after wound débridement. **(C)** The same site after surgical débridement down to viable tissue.

Wounds with less tissue loss can be managed conservatively with the same outcome.[82] Because many extravasation injuries occur on the hands, scar management and return of function remain a problem. Proper administration through the correct needle size (small), vein size (large), and dilution of the medication is best. Infusion should be performed as slowly as possible to allow adequate dilution into the blood. Any complaints of pain during infusion warrant immediate cessation of the infusion, assessment of the intravenous site, and adherence to treatment protocols for extravasation as deemed necessary. Calcium gluconate is less likely to extravasate and should be used instead of calcium chloride for the management of low serum levels, especially those levels that are not life threatening. Because many of these cases may elicit external review and sometimes legal review, documentation is crucial to determine what care was given prior to and after the medication was given. Nurses need to be especially vigilant when administering medications prone to extravasation.

Patient Teaching

Patients don't always give a complete history or believe that certain information is relevant to a clinician pertaining to their wound. Because certain diseases or treatments travel to certain locations, or even contact with certain insects and animals can be associated

with some atypical wounds, telling a clinician about these factors can be important in determining the correct etiology of the wound. Instruct patients to always tell the clinician if they:

- have inflammatory bowel disease, as this is often an underlying etiology in pyoderma gangrenosum
- lived in a developing country, as there could be a risk for Buruli ulcers
- visited or lived in a tropical region of Central or South America, as deep fungal infections, systemic mycoses, or exposure to soil (such as being on a farm in a subtropical location, especially in South America) could lead to chromoblastomycosis or paracoccidioidomycosis
- have eaten raw oysters or been fishing or participated in water activities in Atlantic Coast waters, as this can lead to *V. vulnificus* infection
- have a history of chronic renal failure and/or are on hemodialysis, as calcific uremic arteriopathy ulcers (calciphylaxis) can occur
- have an impaired immune system, such as from HIV or alcoholism, as sporotrichosis may be present
- take certain medications, such as warfarin (Coumadin), as this can cause necrosis.

Summary

Tissue biopsy is often the first step in diagnosing an atypical wound. Treating the underlying cause, when possible, is the initial step in caring for patients with atypical wounds. Anti-infective agents may be used for infectious ulcers, malignancies may require surgical removal, and anti-inflammatory agents may be used for inflammatory ulcers. In addition, the use of a moist healing environment, compression dressings (in the absence of arterial insufficiency) for leg lesions, off-loading areas at risk for prolonged pressure, and maximizing patients' nutritional status are essential.

Despite these measures, healing is often slow in patients with atypical wounds, and multiple treatment modalities may be needed. Prolonged healing leads to increased morbidity and decreased quality of life as well as an increase in direct and indirect costs of care. Adjunctive therapies are often used, aimed at both increasing the number of patients who will heal (effectiveness of therapy) and the speed at which they heal (cost-effectiveness of therapy).

PATIENT SCENARIO

Clinical Data

Mr. JM is a 43-year-old patient who presents to your clinic for the first time for treatment of a painful ulcer. He has had the ulcer for approximately 2 months. He states that it started as a small pustule on his upper back that has since opened into a very tender ulcer. Two months ago, he saw his primary care doctor. The doctor told him the wound appeared infected and needed to be débrided; he would also need antibiotics. Within the week after débridement, the wound increased in size. Upon further questioning, you find out that Mr. JM has a history of cancer. On physical exam, the ulcer is approximately 6 cm in diameter with irregular, violaceous, undermined borders. The base of the ulcer is purulent with ample

exudate present. The patient experiences extreme pain when the surrounding area is palpated.

Case Discussion

Several aspects of the history and physical exam should clue you in to the correct diagnosis of pyoderma gangrenosum. Pyoderma gangrenosum classically starts as a pustule that ulcerates and enlarges, although not all ulcers start this way. The appearance of the wound is also classic, with violaceous, undermined borders with a purulent base.

The patient's past medical history is very important in this case. Nearly 50% of patients with pyoderma gangrenosum have an underlying disease, the most common of which is inflammatory bowel disease. Other associated diseases include malignancies and inflammatory arthritis.

Mr. JM clearly demonstrates pathergy, the development of ulcers in an area of trauma or worsening of the ulcer with trauma; in this case, it was evident by the worsening of the ulcer following débridement. Other patients may report the development of ulcers following seemingly trivial trauma, such as hitting one's leg against a coffee table.

Treatment consisted of cleansing the wound with normal saline and dressing the wound with an absorbent dressing. High-dose systemic steroids were started, and the ulcer gradually resolved over the next 6 weeks, at which point the steroids were tapered.

Show What You Know

1. **Which of the following is the most important reason to recognize a wound as atypical?**
    A. Atypical wounds take longer to heal.
    B. Treatment varies based on etiology.
    C. Standard wound healing therapies do not apply.
    D. Atypical wounds are more painful than standard wounds.

2. **Which of the following may be performed on tissue biopsies of wounds?**
    A. Histology
    B. Culture
    C. Immunofluorescence
    D. All of the above

3. **Which of the following should not typically be débrided?**
    A. Diabetic foot ulcer
    B. Ulcers due to infectious causes
    C. Pyoderma gangrenosum ulcers
    D. Ulcers due to vasculitis

4. **Sporotrichosis is a fungal infection caused by:**
    A. *S. schenckii.*
    B. *M. ulcerans.*
    C. *F. pedrosoi.*
    D. *M. marinum.*

5. **Wounds caused by cryofibrinogenemia are classified as a:**
    A. *mycobacterium.*
    B. pyoderma gangrenosum.
    C. vasculopathy.
    D. metabolic disease.

6. **Which type of wound is a rare, often fatal condition characterized by progressive cutaneous necrosis that**

occurs in patients with end-stage renal disease?

A. Calciphylaxis
B. Vasculopathy
C. Radiation dermatitis
D. Chemical burn

7. Factitial dermatitis is a:

A. rare condition.
B. self-imposed injury.
C. red rash.
D. dry, scaly scab.

8. Wounds caused by vasculitis are due to:

A. thrombosis.
B. vascular inflammation.
C. extravasation.
D. venous insufficiency.

9. Wounds caused by vasculopathy are due to:

A. thrombosis.
B. vascular inflammation.
C. extravasation.
D. venous insufficiency.

Answers to these questions are found on page 687.

References

1. Sen, C.K., Gordillo, G.M., Roy, S., et al. “Human Skin Wounds: A Major and Snowballing Threat to Public Health and the Economy.” Wound Repair and Regeneration 17:763-71, 2009.
2. European Wound Management Association. Atypical Wounds. 2019. Available at: http://ewma.org/what-we-do/ewma-projects
3. Patel, G.K., Grey, J.E., Harding, K.G. “Uncommon Causes of Ulceration.” British Medical Journal 332(7541):594-6, 2006.
4. Carlson, J.A. “The Histological Assessment of Cutaneous Vasculitis.” Histopathology 56(1):3-23, 2010.
5. Marzano, A.V., Vezzoli, P., Berti, E. “Skin Involvement in Cutaneous and Systemic Vasculitis.” Autoimmunity Reviews 12(4):467-76, February 2013.
6. Chen, K.R., Carlson, J.A. “Clinical Approach to Cutaneous Vasculitis.” American Journal of Clinical Dermatology 9(2):71-92, 2008.
7. Carlson, J.A., Ng, B.T., Chen, K.R. “Cutaneous Vasculitis Update: Diagnostic Criteria, Classification, Epidemiology, Etiology, Pathogenesis, Evaluation and Prognosis.” American Journal of Dermatopathology 27(6):504-28, 2005.
8. Lapraik, C., Watts, R., Scott, D.G. “Modern Management of Primary Systemic Vasculitis.” Clinical Medicine 7(1):43-7, 2007.
9. Callen, J.P., Jackson, J.M. “Pyoderma Gangrenosum: An Update.” Rheumatic Diseases Clinics of North America 33(4):787-802, 2007.
10. Alavi, A., French, L.E., Davis, M.D., et al. “Pyoderma Gangrenosum: An Update on Pathophysiology, Diagnosis and Treatment.” American Journal of Clinical Dermatology, 18:355-72, 2017.
11. Jockenhofer, F., Wollina, U., Salva, K.A., et al. “The PARACELSUS Score: A Novel Diagnostic Tool for Pyoderma Gangrenosum.” British Journal of Dermatology 180:615-20, 2019.
12. Bennett, M.L., Jackson, J.M., Jorizzo, J.L., et al. “Pyoderma Gangrenosum. A Comparison of Typical and Atypical Forms with an Emphasis on Time to Remission. Case Review of 86 Patients from 2 Institutions.” Medicine (Baltimore) 79(1):37-46, 2000.
13. Sheldon, D.G., Sawchuk, L.L., Kozarek, R.A., et al. “Twenty Cases of Peristomal Pyoderma Gangrenosum: Diagnostic Implications and Management.” Archives of Surgery 135(5):564-8; discussion 68-9, 2000.
14. Blitz, N.M., Rudikoff, D. “Pyoderma Gangrenosum.” Mount Sinai Journal of Medicine 68:287-97, 2001.
15. Ruocco, E., Sangiuliano, S., Gravina, A.G., et al. “Pyoderma Gangrenosum: An Updated Review.” Journal of the European Academy of Dermatology and Venereology 23(9):1008-17, 2009.
16. Brooklyn, T.N., Dunnill, M.G., Shetty, A., et al. “Infliximab for the Treatment of Pyoderma Gangrenosum: A Randomised, Double Blind, Placebo Controlled

Trial." Gut 55(4):505-9, April 2006.
17. Sadati, M.S., Dastgheib, L., Aflaki, E. "Recalcitrant Cases of Pyoderma Gangrenosum, Responding Dramatically to Systemic Tacrolimus." Giornale Italiano di Dermatologia e Venereologia 152:308-10, 2017.
18. Din, R.S., Tsiaras, W.G. Li, D.G., et al. "Efficacy of Systemic Dapsone Treatment for Pyoderma Gangrenosum: A Retrospective Review." Journal of Drugs in Dermatology, 17:1058-60, 2018.
19. von Laffert, M., Stadie, V., Wohlrab, J., et al. "Hidradenitis Suppurativa/Acne Inversa: Bilocated Epithelial Hyperplasia with Very Different Sequelae." British Journal of Dermatology 164:367-71, 2011.
20. Onderdijk, A.J., van der Zee, H.H., Esmann, S., et al. "Depression in Patients with Hidradenitis Suppurativa." Journal of the European Academy of Dermatology and Venereology 27:473-8, 2013.
21. Nazzaro, G., Passoni, E., Muratori, S. et al. "Comparison of Clinical and Sonographic Scores in Hidradenitis Suppurativa and Proposal of a Novel Ultrasound Scoring System." Giornale Italiano di Dermatologia e Venereologia 2018. doi: 10.23736/S0392-0488.18.06196-5. [Epub ahead of print.]
22. Groves, R. "Unusual Cutaneous Mycobacterial Diseases." Clinics in Dermatology 13(3):257-63, 1995.
23. Dodiuk-Gad, R., Dyachenko, P., Ziv, M., et al. "Nontuberculous Mycobacterial Infections of the Skin: A Retrospective Study of 25 Cases." Journal of the American Academy of Dermatology 57(3):413-20, 2007.
24. Kwyer, T.A., Ampadu, E. "Buruli Ulcers: An Emerging Health Problem in Ghana." Advances in Skin & Wound Care 19(9):479-86, 2006.
25. Ampadu, E., Kwyer, T.A., Otcher, Y. "Buruli Ulcer: Picture of an Emerging Health Challenge and the Response in Ghana." WCET Journal 26(4):30-6, 2006.
26. Sarfo, F.S., Phillips, R., Asiedu, K., et al. "Clinical Efficacy of Combination of Rifampin and Streptomycin for Treatment of *Mycobacterium ulcerans* Disease." Antimicrobial Agents and Chemotherapy 54(9):3678-85, September 2010.
27. Phillips, R.O., Sarfo, F.S., Abass, M.K., et al. "Clinical and Bacteriological Efficacy of Rifampin-Streptomycin Combination for Two Weeks Followed by Rifampin and Clarithromycin for Six Weeks for Treatment of *Mycobacterium ulcerans* Disease." Antimicrobial Agents and Chemotherapy 58(2):1161-6, February 2014.
28. Treatment of *Mycobacterium ulcerans* Disease (BURULI ULCER). 2012. Available at: https://www.who.int/iris/bitstream/10665/77771/1/9789241503402_eng.pdf?ua=1
29. Lupi, O., Tyring, S.K., McGinnis, M.R. "Tropical Dermatology: Fungal Tropical Diseases." Journal of the American Academy of Dermatology 53(6):931-51,

quiz 52-4, 2005.
30. Ramos-e-Silva, M., Vasconcelos, C., Carneiro, S. “Sporotrichosis.” Clinics in Dermatology 25(2):181-7, 2007.
31. Lopez Martinez, R., Mendez Tovar, L.J. “Chromoblastomycosis.” Clinics in Dermatology 25(2):188-94, 2007.
32. Torres-Guerrero, E., Isa-Isa, R., Isa, M., et al. “Chromoblastomycosis.” Clinics in Dermatology 30(4):403-8, July-August 2012.
33. Negroni, R., Tobon, A., Bustamante, B., et al. “Posaconazole Treatment of Refractory Eumycetoma and Chromoblastomycosis.” Revista do Instituto de Medicina Tropical de São Paulo 47(6):339-46, 2005.
34. Ramos, E.S.M., Saraiva Ldo, E. “Paracoccidioidomycosis.” Dermatologic Clinics 26(2):257-69, vii, 2008.
35. Lichon, V., Khachemoune, A. “Mycetoma: A Review.” American Journal of Clinical Dermatology 7(5):315-21, 2006.
36. Fahal, A.H., Sheik, H.E., Homeida, M.M., et al. “Ultrasonographic Imaging of Mycetoma.” The British Journal of Surgery 84(8):1120-2, 1997.
37. Ameen, M., Arenas, R. “Developments in the Management of Mycetomas.” Clinical and Experimental Dermatology 34(1):1-7, 2009.
38. Henao-Martinez, A.F., Beckham, J.D. “Cryptococcosis in Solid Organ Transplant Recipients.” Current Opinion in Infectious Diseases 28:300-7, 2015.
39. Gavalda, J., Meije, Y., Fortun, J., et al. “Invasive Fungal Infections in Solid Organ Transplant Recipients.” Clinical Microbiology and Infection 20(Suppl 7):27-48, 2014.
40. Chowdhary, A., Meis, J.F., Guarro, J., et al. “ESCMID and ECMM Joint Clinical Guidelines for the Diagnosis and Management of Systemic Phaeohyphomycosis: Diseases Caused by Black Fungi.” Clinical Microbiology and Infection 20(Suppl 3):47-75, 2014.
41. de Araujo, M.R., Aquino, C., Scaramal, E., et al. “*Vibrio vulnificus* Infection in Sao Paulo, Brazil: Case Report and Literature Review.” Brazilian Journal of Infectious Diseases 11(2):302-5, 2007.
42. Brennt, C.E., Wright, A.C., Dutta, S.K., et al. “Growth of *Vibrio vulnificus* in serum from alcoholics: association with high transferrin iron saturation.” Journal of Infectious Diseases 164:1030-2, 1991.
43. Bross, M.H., Soch, K., Morales, R., et al. “*Vibrio vulnificus* Infection: Diagnosis and Treatment.” American Family Physician 76(4):539-44, 2007.
44. Blake, P.A., Merson, M.H., Weaver, R.E., et al. “Disease Caused by a Marine Vibrio. Clinical Characteristics and Epidemiology.” New England Journal of Medicine 300:1-5, 1979.
45. Salcido, R.S. “Necrotizing Fasciitis: Reviewing the Causes and Treatment Strategies.” Advances in Skin & Wound Care 20(5):288-93, quiz 94-5, 2007.
46. McHenry, C.R., Piotrowski, J.J., Petrinic, D., et al. “Determinants of Mortality for Necrotizing Soft-tissue Infections.” Annals of Surgery 221(5):558-63;

discussion 63-5, 1995.
47. Ogilvie, C.M., Miclau, T. “Necrotizing Soft Tissue Infections of the Extremities and Back.” Clinical Orthopaedics and Related Research 447:179-86, 2006.
48. Bellapianta, J.M., Ljungquist, K., Tobin, E., et al. “Necrotizing Fasciitis.” The Journal of the American Academy of Orthopaedic Surgeons 17(3):174-82, 2009.
49. Unalp, H.R., Kamer, E., Derici, H., et al. “Fourniers Gangrene: Evaluation of 68 Patients and Analysis of Prognostic Variables.” Journal of Postgraduate Medicine 54(2):102-5, 2008.
50. Angoules, A.G., Kontakis, G., Drakoulakis, E., et al. “Necrotising Fasciitis of Upper and Lower Limb: A Systematic Review.” Injury 38(Suppl 5):S19-26, 2007.
51. Saadoun, D., Elalamy, I., Ghillani-Dalbin, P., et al. “Cryofibrinogenemia: New Insights into Clinical and Pathogenic Features.” American Journal of Medicine 122(12):1128-35, 2009.
52. Falanga, V., Kirsner, R.S., Eaglstein, W.H., et al. “Stanozolol in Treatment of Leg Ulcers due to Cryofibrinogenaemia.” Lancet 338(8763):347-8, 1991.
53. Tedeschi, A., Barate, C., Minola, E., et al. “Cryoglobulinemia.” Blood Reviews 21(4):183-200, 2007.
54. Ferri, C. “Mixed Cryoglobulinemia.” Orphanet Journal of Rare Diseases 3:25, 2008.
55. Pawlotsky, J.M. “New Hepatitis C Therapies: The Toolbox, Strategies, and Challenges.” Gastroenterology 146(5):1176-92, 2014.
56. Iannuzzella, F., Vaglio, A., Garini, G. “Management of Hepatitis C Virus-Related Mixed Cryoglobulinemia.” American Journal of Medicine 123(5):400-8, 2010.
57. Sharma, P.V., Babu, S.C., Shah, P.M., et al. “Changing Patterns of Atheroembolism.” Cardiovascular Surgery 4:573-9, 1996.
58. Lansberg, M.G., O’Donnell, M.J., Khatri, P., et al. “Antithrombotic and Thrombolytic Therapy for Ischemic Stroke: Antithrombotic Therapy and Prevention of Thrombosis, 9th ed: American College of Chest Physicians Evidence-Based Clinical Practice Guidelines.” Chest 141: e601S-36S, 2012.
59. Eby, C. “Antiphospholipid Syndrome Review.” Clinics in Laboratory Medicine 29(2):305-19, 2009.
60. Lim, W. “Antiphospholipid Antibody Syndrome.” Hematology/the Education Program of the American Society of Hematology. *Education Program* 233-9, 2009.
61. Andersen, R.J., Campoli, J., Johar, S.K., et al. “Suspected Brown Recluse Envenomation: A Case Report and Review of Different Treatment Modalities.” The Journal of Emergency Medicine 41(2):e31-7, 2011.
62. Rand, J.H., Wu, X.X., Quinn, A.S., et al. “The Annexin A5-Mediated Pathogenic Mechanism in the Antiphospholipid Syndrome: Role in Pregnancy Losses and Thrombosis.” Lupus 19(4):460-9, 2010.

Dauden, E., Onate, M.J. “Calciphylaxis.” Dermatologic Clinics 26(4):557-68, ix, 2008.
64. Guldbakke, K.K., Khachemoune, A. “Calciphylaxis.” International Journal of Dermatology 46(3):231-8, 2007.
65. Budisavljevic, M.N., Cheek, D., Ploth, D.W. “Calciphylaxis in Chronic Renal Failure.” Journal of the American Society of Nephrology 7(7):978-82, 1996.
66. Anderson, K.G. “Calcific Uremic Arteriolopathy: Overview for the Nurse.” AACN Advanced Critical Care 24(3):285-300, 2013.
67. Weenig, R.H., Sewell, L.D., Davis, M.D., et al. “Calciphylaxis: Natural History, Risk Factor Analysis, and Outcome.” Journal of the American Academy of Dermatology 56(4):569-79, 2007.
68. Fine, A., Zacharias, J. “Calciphylaxis Is Usually Non-Ulcerating: Risk Factors, Outcome and Therapy.” Kidney International 61(6):2210-7, 2002.
69. Selye, H., Gabbiani, G., Strebel, R. “Sensitization to Calciphylaxis by Endogenous Parathyroid Hormone.” Endocrinology 71:554-8, 1962.
70. Rogers, N.M., Coates, P.T. “Calcific Uraemic Arteriolopathy: An Update.” Current Opinion in Nephrology and Hypertension 17(6):629-34, 2008.
71. Weenig, R.H. “Pathogenesis of Calciphylaxis: Hans Selye to Nuclear Factor Kappa-B.” Journal of the American Academy of Dermatology 58(3):458-71, 2008.
72. Yang, D., Morrison, B.D., Vandongen, Y.K., et al. “Malignancy in Chronic Leg Ulcers.” Medical Journal of Australia 164(12):718-20, 1996.
73. Copcu, E. “Marjolin’s Ulcer: A Preventable Complication of Burns?” Plastic and Reconstructive Surgery 124(1):156e-64e, 2009.
74. Enoch, S., Miller, D.R., Price, P.E., et al. “Early Diagnosis Is Vital in the Management of Squamous Cell Carcinomas Associated with Chronic Non Healing Ulcers: A Case Series and Review of the Literature.” International Wound Journal 1(3):165-75, 2004.
75. Chang, A., Spencer, J.M., Kirsner, R.S. “Squamous Cell Carcinoma Arising from a Nonhealing Wound and Osteomyelitis Treated with Mohs Micrographic Surgery: A Case Study.” Ostomy/Wound Management 44(4):26-30, 1998.
76. Wilson, D.C., King, L.E. Jr. “Spiders and Spider Bites.” Dermatologic Clinics 8:277-86, 1990.
77. Hogan, C.J., Barbaro, K.C., Winkel, K. “Loxoscelism: Old Obstacles, New Directions.” Annals of Emergency Medicine 44(6):608-24, 2004.
78. Palao, R., Monge, I., Ruiz, M., et al. “Chemical Burns: Pathophysiology and Treatment.” Burns 36(3):295-304, 2010.
79. Radvansky, L.J., Pace, M.B., Siddiqui, A. “Prevention and Management of Radiation-Induced Dermatitis, Mucositis, and Xerostomia.” American Journal of Health-System Pharmacy 70(12):1025-32, 2013.
80. Nazarian, R.M., Van Cott, E.M., Zembowicz, A., et al. “Warfarin-Induced Skin Necrosis.” Journal of the American Academy of Dermatology 61(2):325-32,

2009.
81. Cheng, A., Scheinfeld, N.S., McDowell, B., et al. “Warfarin Skin Necrosis in a Postpartum Woman with Protein S Deficiency.” Obstetrics and Gynecology 90(4 Pt 2):671-2, 1997.
82. Doellman, D., Hadaway, L., Bowe-Geddes, L.A., et al. “Infiltration and Extravasation: Update on Prevention, and Management.” Journal of Infusion Nursing 32(4):203-11, 2009.

23

Spinal Cord Injury Population

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Objectives

After reviewing this chapter, you will be able to:

- describe successful strategies for reducing the incidence of pressure injuries in the person with an spinal cord injury (SCI)
- identify risk factors for the development of pressure injuries throughout the life of a patient with an SCI
- discuss the major health complications of a person with an SCI.

This chapter presents information on the effects of pressure injuries in the spinal cord injury (SCI) population. The person with an SCI faces a lifelong risk for the development of pressure injuries. Although often preventable, in this population, pressure injuries affect every aspect of a person's life from achieving rehabilitation goals, independence, and participation, and pursuing educational, vocational, and leisure activities. Better assessment measures, as well as, new technology in wheelchairs, seating, beds, mattresses, surgical techniques and dressings have enabled clinicians to identify SCI persons at highest risk for pressure injuries. Some of these innovations have resulted in effective prevention strategies that can be incorporated into their lifestyles.

Spinal Cord Injury Incidence and Prevalence

Approximately 282,000 persons are living with SCI in the United States today, of which 17% (42,000) are veterans.[1,2] Incidence is 17,000 new cases of SCI each year based on an estimate of 54 cases of SCI per million population in the United States.[2] The most recent data on the demographics of SCI are derived from the National Spinal Cord Injury Statistical Center (NSCISC).[2] The NSCISC provides statistics on five major categories of etiology:

- Motor vehicle crashes 38.0%
- Falls 30.5%
- Acts of violence 13.5%
- Recreational sporting activities 9.0%
- Medical/surgical 5%
- Causes that do not fit into any of these categories 4.0%[2]

SCI occurs most frequently in males aged 16 to 30 (55%), and males make up more than 80% of the total population of persons with SCI.[2] The average age at injury today is 42.0 years (up from 29 years at the end of the 1970s). Changes in race have been observed since 2010. Currently, 63.5% are non-Hispanic White, 22% are non-Hispanic Black, 11% are Hispanic, 2% are Asian, and 0.4% other. This may be due, in part, to the trends in the general population of the United States. Over half of the SCI population had completed high school at the time of injury. More than half are single. Although more than half were employed at the time of injury, only 12% are employed 1 year following SCI.[2] By 20 years postinjury, almost one-third are employed.

People with SCI are at risk for a number of secondary medical complications. For instance, pulmonary complications are among the most common cause of death during both the acute and chronic phases after SCI.[3] Other potential complications arising soon after injury—some of which may become lifelong problems—include pressure injuries, urinary tract infections (UTIs), osteoporosis, fractures, autonomic dysreflexia, and heterotopic ossification.

Pressure injuries are among the most common long-term secondary medical complications observed at annual follow-up visits.[4] As such, they are a serious, costly, and potentially life-threatening complication of SCI. Clinical observations and research studies have confirmed staggering costs and human suffering, including a profoundly negative impact on the person's general physical health, socialization, financial status, and body image, compounded by a loss of independence and control.[5]

Reliable and current data on the incidence and prevalence of pressure injuries in the person with SCI have been difficult to obtain, primarily because there were variations in the data collection methods, limiting standardization of the statistics. These limitations include the use of different classification systems to stage pressure injuries, the inability to compare varied populations (e.g., acute or chronic, traumatic or nontraumatic SCI) presenting with or developing pressure injuries, and the use of different methods of obtaining data, such as direct observation or retrospective chart review.[6] However, reported prevalence rates of pressure injuries for persons with SCI residing in the community have ranged from 17% to 33%.[7]

The number of pressure injuries increases significantly with duration of injury.[8] For persons with SCI, the incidence rate increased from 15% at 1 year postinjury to nearly 30% at 20 years postinjury.[9] This may be the result of a combined effect of aging and long-term SCI. Aging with SCI has been shown to negatively impact many health factors including the odds ratio for pressure injury development.[10]

Scope of the Problem

The database of the Spinal Cord Injury Model Systems is one of the most reliable resources from which to obtain data that reflect the scope of the problem. The National Institute on Disability, Independent Living, and Rehabilitation Research (NIDILRR) sponsors the Model Systems Program, a federal extramural grant program of selected research and demonstration sites. Model Systems sites provide exemplary, state-of-the-art care from the time of injury through acute medical care, comprehensive rehabilitation, and long-term follow-up and health maintenance services.[2] An individual is included in the database only if he or she was admitted to a system facility within 24 hours of trauma. Although the Model Systems database has included statistics on pressure injuries in patients with SCI since 1981, these statistics do not appear every year in the Facts and Figures published data set.

An increased prevalence of pressure injuries is seen in persons with longer SCI durations, especially among those 10 years or more postinjury.[11] Among veterans with SCI, over one-third report having pressure injuries during the previous year.[12] Pressure injuries remain the second leading cause of rehospitalization after urinary problems for persons with SCI enrolled in the U.S. SCI Model Systems database. Pressure injuries account for 17% of the rehospitalizations in the first year after injury, 23% at 5 years, 29% at 10 years, and 38% at 20 years postinjury.[2]

In a study by Brienza et al., 37.5% of the SCI participants developed at least one pressure injury during acute care hospitalization and inpatient rehabilitation. There was a significant association of pressure injury incidence for those with pneumonia and mechanical ventilation ($P = 0.01$) and higher injury severity-ASIA A ($P = 0.01$).[11] Model Systems data indicate that 34% of patients admitted to a Model Systems facility within 24 hours of an SCI developed at least one pressure injury during acute care or rehabilitation, with prevalence increasing over time following injury.[13] On follow-up, 15% had a pressure injury at their first annual examination, 20% at year 5,

23% at year 10, 24% at year 15, and 29% at year 20. These numbers are based on 4,065 patients, 2,971 of whom developed pressure injuries. Other investigators have reported prevalence rates ranging from 17% to 33% in populations of SCI patients residing in the community.[7,14] The occurrence of pressure injuries is among the most common long-term secondary medical complications identified during annual follow-up visits in individuals with paraplegia (T1 to S5, neurological level), who are more likely to be rehospitalized for pressure injuries.[2,9,15] Risk factors include complete injury (classified as A, B, or C by the American Spinal Injury Association to denote complete vs. incomplete injury), tetraplegia, older age, comorbidities, and violent injury[9] (Table 23-1).

Table 23-1 American Spinal Injury Association (ASIA) Impairment Scale

A—Complete: No motor or sensory function is preserved in the sacral segments S4–S5.
B—Incomplete: Sensory but not motor function is preserved below the neurological level and includes the sacral segments S4–S5.
C—Incomplete: Motor function is preserved below the neurological level, and more than half of key muscles below the neurological level have a muscle grade <3.
D—Incomplete: Motor function is preserved below the neurological level, and at least half of key muscles below the neurological level have a muscle grade of 3.
E—Normal: Motor and sensory functions are normal.

Recurrence Rate

High rates of pressure injury recurrence also have been reported, ranging from 21% to 79% regardless of treatment.[16,17] Epidemiological studies have found that 36% to 50% of all patients with SCI who develop pressure injuries will develop a recurrence within the first year after initial healing.[7,14,16–18] Niazi et al.[16] reported a recurrence rate of 35% regardless of the type of treatment (medical or surgical) provided. Holmes et al.[19] found that 55% of their sample, most of whom had a history of severe previous injuries, experienced a recurrence within 2 years after surgical repair. In a 20-year study in Canada (1976–1996), Schryvers et al.[20] studied 168 SCI patients admitted 415 times for treatment of 598 severe recurring pressure injuries. Of these recurrences, 31% (185) occurred at the same site as the previous ulcer and 21% (125 ulcers) occurred at a different site. Goodman et al.[21] observed a recurrence or new pressure injury development rate of 79% within a 1- to 6-year follow-up time frame.

Some investigators found that pressure injury history is a more viable measure of pressure injury outcome than measures taken at any single point in time over a brief period. Other studies reveal the protective mechanisms against pressure injury recurrence. For example, Krause and Broderick[22] reported that 13% of their sample of 633 subjects had recurring pressure injuries (one or more per year) over a 5-year period. Their findings suggested that lifestyle, exercise, and diet were protective mechanisms against pressure injury recurrence.

Pressure injury recurrence in the person with an SCI has been associated with gender (male), age (older), ethnicity (African American), unemployment, residence in a nursing home, and previous pressure injury surgery.[22,23] Most of the literature describing recurrence following surgery focuses on surgical techniques.[24–28] Investigators have reported recurrence rates of 11% to 29% in patients with postoperative complications and 6% to 61% in patients without postoperative complications.[24–30] Relander and Palmer[28] recommended that social factors be studied to determine the causes

of pressure injury recurrence after surgical repair and suggested that patients who do not display the appropriate knowledge regarding pressure injury prevention should be counseled before consideration for surgery. Disa et al.[24] reported that high recurrence rates among patients with traumatic paraplegia were associated with substance abuse and the absence of an adequate social support system. They suggested developing more effective educational programs for both patients and caregivers.[24] In other studies, Mandrekas and Mastorakos[29] and Rubayi et al.[30] reported that inadequate patient education with regard to pressure injury prevention contributed to the recurrence rates.

Recurrence also is a significant problem for veterans with SCI. Guihan et al.[31] reported that there are four significant predictors of pressure injury recurrence in veterans. They are race (being black), more comorbidities indicating a higher burden of illness, Salzberg Pressure Ulcer (PrU) Risk Assessment Scale score (which measures 15 items associated with SCI, cardiovascular and pulmonary disease, albumin and hematocrit, and impaired cognition), and longer sitting time at discharge from the hospital following the treatment of a pressure injury.[31]

Financial Concerns

The financial burden of pressure injuries is undoubtedly immense, although estimates of the cost of preventing and treating pressure injuries in the patient with an SCI are not easily verified. Between 2008 and 2012, the medical management of pressure injuries costs the U.S. healthcare system $9.1 to $11.6 billion per year. The median total hospital cost for persons with pressure injuries was $36,500 (mean $72,000 ± $122,900) compared to persons without pressure injures, whose median total hospital cost was $17,200 (mean $32,200 ± $57,000).[32,33] Although these data were not specific to persons with SCI, one could extrapolate from these data the financial implications of pressure injuries for patients with SCI. The financial burden does not begin to reflect the personal and social costs experienced by patients and their families. These include loss of independence and self-esteem; time away from work, school, or family; and, ultimately, diminished quality of life. It is well documented that the development of pressure injuries increases both hospital costs and length of stay for all patients.[34]

Throughout their lifetime, persons with SCI are at risk for the development of pressure injuries. Most of the reported costs are associated with various treatment interventions, especially dressings.[35] In 2003, Garber and Rintala[36] reported that of 102 veterans' charts reviewed, a total of 625 visits were made to treat 400 pressure injuries at a cost of $250 per outpatient visit (approximate costs from 1998).[37] Stage 4 was the most prevalent pressure injury seen, and pelvic ulcers accounted for almost two-thirds of the worst wounds reported. The average number of clinic or home visits for pressure injury treatment was more than six per person. More than half of the study sample was admitted to the hospital for pressure injury treatment at least once during the study period. The average number of hospital admissions was two; almost 30% of veterans were admitted three times or more.[36]

Two reports examined the direct costs of the treatment of pressure injuries in persons with SCI. Chan et al.[38] studied the costs of treating

chronic pressure injuries among community-dwelling SCI individuals in Ontario, Canada. The average monthly cost was found to be $4,745 per community-dwelling SCI individual. Costs for hospital admission were found to make up for the largest percentage of the total costs.[38] In 2011, Stroupe et al. studied the costs of treating pressure injuries among veterans with SCI.[39] The total inpatient cost per year for SCI veterans with pressure injuries was $91,341, while that for a veteran without pressure injury was $13,754. The total outpatient cost per year for SCI veterans with pressure injuries was $19,844, compared to $11,829 for those without pressure injuries.[39]

Total healthcare costs for individuals with SCI are higher than other groups at risk for pressure injuries development and increasing rapidly.[40] The additional costs of treating veterans with SCI who have pressure ulcers have been estimated to be over $73,000 per annum due primarily to higher inpatient costs.[39]

Risk Factors

More than 200 risk factors for the development of pressure injuries have been reported in the literature. Most of the risk factors were derived from studying elderly nursing home residents. However, many of the risk factors for these patients differ from those experienced by patients with SCI. Immobility increases the risk of pressure injury development in both populations. Unlike nursing home residents, however, patients with SCI are encouraged to oversee or direct their own daily care and are expected to take primary responsibility for pressure injury prevention. As a result of the limitations imposed by the variables among studies, the literature is often contradictory regarding the effects of a particular risk factor or set of factors potentially responsible for the development of pressure injuries. Different populations (e.g., acute or chronic, traumatic or nontraumatic SCI patients), inadequate sample sizes, different ways of standardizing the dependent measures, and poor or uncontrolled study designs all add confusion to the interpretation of study results.[41,42]

According to Chen et al., the risk of pressure injuries seems to be steady in the first 10 years but increases 15 years postinjury.[43] Males, the elderly, blacks, singles, individuals who have less than a high school education, the unemployed, persons with complete SCI, and those with a history of pressure injuries, rehospitalization, nursing home stay, and other medical comorbidities are at greater risk of developing pressure injuries.[43] Although the number of days hospitalized and frequency of rehospitalizations decreased, the number of pressure injuries increased over time.[43] Charlifue et al.[8] found that although the number of pressure injuries increased as time passed, the best predictor of pressure injury over time was a previous history of pressure injuries.

Despite these limitations, a number of pressure injury risk factors specific to the patient with SCI have been identified and described in the literature. Byrne and Salzberg[41] summarized the major pressure injury risk factors for the patient with an SCI as follows:

- Severity of the SCI (immobility, completeness of the injury, urinary incontinence, diminished or absent sensation, and severe spasticity)
- Preexisting conditions (advanced age, smoking, lung and cardiac disease, diabetes and renal disease, and impaired cognition)
- Residence in a nursing home
- Malnutrition and anemia

Characteristics associated with better pressure injury outcomes include maintaining normal weight, returning to work and family roles, and not smoking or having a history of tobacco use, suicidal behavior, incarceration, or alcohol or drug abuse.[44] The Consortium for Spinal Cord Medicine Clinical Practice Guidelines "Pressure Ulcer Prevention and Treatment Following Spinal Cord Injury: A Clinical Practice Guideline for Health-Care Professionals"[6] categorized pressure injury risk factors in patients with SCI as follows:

- Demographic factors (age, gender, ethnicity, marital status, and education)
- Physical-, medical-, and SCI-related factors (level and completeness of the injury; activity and mobility; nutrition, bladder, bowel, and moisture control; and comorbidities, such as diabetes and spasticity)
- Psychological and social factors (psychological distress, financial problems, cognition, substance abuse, adherence to recommended prevention behaviors/strategies, context and health beliefs and practices)
- Support surfaces for bed and wheelchair

In 1979, Anderson and Andberg[45] identified psychological factors associated with the development of pressure injuries, including the patient's unwillingness to take responsibility for his or her skin care, low self-esteem, and dissatisfaction with life activities. Gordon et al.[46] also found poor social adjustment in the patient with an SCI and a pressure injury. The Second Edition of the Paralyzed Veterans of America's (PVA) Consortium for Spinal Cord Injury Clinical Practice Guidelines on "Pressure Ulcer Prevention and Treatment Following Spinal Cord Injury: A Clinical Practice Guideline for Health-Care

Professionals"[6] lists the following psychological and cognitive risk factors:

- *Psychological factors*: Depression, anxiety, negative self-concept, poorly managed anger, and frustration may interfere with cooperation between individual and his/her care providers and may be associated with inactivity, self-neglect, and poor medical adherence.[47]
- *Cognitive impairment*: Associated traumatic brain injury and illegal drug use may interfere with cognitive function.[48]

Social, environmental, contextual, and circumstantial factors: In recent years, there have been attempts to identify risk factors, other than obvious medical- and SCI-related ones, within a person's life that contribute to the development of pressure injuries, interfere with healing, and fail to prevent recurrence. Homelessness, gang membership, financial problems, and lack of social support have been shown to cause frequent disruptions, promote participation in high-risk activities, and may put the person in a state of danger. The lack of access to care, services, and supports can indeed influence the development of pressure injuries.[49–52] In persons with chronic SCI and pressure injuries, it is important to assess the individual's motivation to stay ulcer-free.[51,52] Motivation drives the daily actions or inactions of the individual. Lack of motivation to perform preventative activities may be associated with depression, lack of social support, or poverty. Conflicting motivations, such as wanting to stay fully engaged in either vocational or recreational pursuits, may interfere with maintaining a strict regimen of pressure injury prevention. Understanding life context, daily routines, and central daily activities should result in a balance between one's daily occupations and pressure injury preventative regimens. Therefore, in order to most effectively prevent and treat pressure injuries in any individual, it is important to not just evaluate the usual risk factors as described throughout the PVA guideline but also to evaluate the context in which the individual with SCI lives, for, if these other factors are not addressed as well, any intervention is not likely to be successful.

Support surfaces for bed and wheelchair: Support surfaces that are old, worn-out, or inappropriate may put the person at risk for

pressure injuries. This topic will be covered in detail in Chapter 11 Pressure Redistribution: Seating, Positioning, and Support Surfaces: the use of both a validated risk assessment tool and clinical judgment to assess risk is strongly recommended. The Salzberg Pressure Ulcer Risk Assessment Form for SCI is only one risk assessment measure validated for persons with SCI.[18] However, in many facilities, the Braden Scale, which is not specific to SCI, is used to determine persons at risk for pressure injuries.

Practice Point

Education: Innovative educational programs are needed to provide persons with SCI with information and the motivation necessary to regain control over their lives. It is necessary to determine how effective these programs are through testing knowledge and observing, monitoring, and documenting preventive behaviors.

Risk-Based Prevention

A person with an SCI is at lifelong risk for the development of pressure injuries including the moment of initial injury. Prolonged immobilization during the hours and days immediately after injury significantly increases the risk. Pressure reduction strategies to protect vulnerable areas of the body should be implemented soon after emergency medical intervention and spinal stabilization.[53]

Prevention strategies must be implemented across the full spectrum of care. A comprehensive preventive plan should be developed for each patient and include information and instructions that are given to patients and their families.[54] (See *Patient Teaching: Preventing Pressure Ulcers in SCI Patients at Home*.)

Printed materials or visual media, such as DVDs, are used frequently to augment formal, informal, one-on-one, or group educational sessions. Some of these materials may be sent home with patients when they are discharged. Because most patient education programs are hospital based, little is known about what information the patient retains, which behaviors or activities are practiced routinely, and the compatibility of the patient's lifestyle with prevention strategies. In the 1970s and 1980s, a number of SCI centers established comprehensive pressure injury prevention education programs.[55,56] Both inpatient and outpatient programs advocated multidisciplinary, coordinated, structured, and wide-ranging approaches to prevention. Some of these programs have been expanded and serve as models for practice today.[54] In a recent study of medically underserved adults with spinal cord injury, investigators identified the following potentially protective factors associated with pressure injuries. Among them are meaningful activity, motivation to prevent negative health outcomes, stability/resources, equipment, communication and self-advocacy skills, personal traits, physical factors, and behaviors/activities.[57] However, some protective factors such as personal traits may be inherent within individuals and unmodifiable.[57] A number of educational needs have been described.

In one study, they have been rank ordered by the authors of the study,[58] including:

- awareness of the lifelong risk for developing pressure injury (including the ability to assess personal risk factors and how risk changes over time)
- ability to take charge of personal skin care regimen and to partner with healthcare providers
- ability to perform prevention strategies consistently that are compatible with level of function and activity and update practices as risk changes
- ability to coordinate social supports.

Effective prevention education and early detection of pressure injuries have been described as critical in managing them in persons with spinal cord injury.[52,54]

The concepts of context and lifestyle as they pertain to pressure injury etiology, prevention, and management have been described in the literature. Clark et al.[51] examined the daily lifestyle influences on the development of pressure injuries among adults with SCI. In-depth interviews and observation were used to obtain detailed descriptive information pertaining to the development of recurring pressure injuries as they relate to participants' overall lifestyle, daily routine, choices, challenges, and prevention strategies. These investigators concluded that prevention is dependent on the uniqueness of an individual's everyday life.[51]

In a qualitative study[52] of 20 adults with SCI and a history of pressure injuries, investigators identified eight multifaceted, interconnected lifestyle principles that explain pressure injury etiology. They include perpetual danger, change/disruption of routine, diminished prevention behaviors, risk ratio of liabilities (risk factors or behaviors) to buffers (factors, behaviors, or people that reduce risk), individualization, presence of prevention awareness and motivation, lifestyle trade-off, and access to needed care, services, and supports.[52]

Patient Teaching*Preventing Pressure Injuries in SCI Patients at Home*

Persons with SCI are at high risk for developing pressure injuries, especially after they return to their homes and communities. The following strategies will help them prevent pressure injuries after they have returned home:

- Perform twice daily comprehensive visual and tactile skin inspection.
- Maintain good personal hygiene.
- Turn and reposition often, and perform frequent weight shifts.
- Use appropriate and well-maintained support surfaces for the bed and wheelchair.
- Maintain adequate nutrition.
- Maintain a healthy lifestyle (avoid alcohol, tobacco, and illegal drugs).

Skin Assessment

The SCI patient with a pressure injury should undergo two assessment phases. The first phase is a comprehensive evaluation and examination, including:

- complete social history including family, financial stability, or other support
- medical history including comorbidities
- physical examination including functional independence (ADLs, mobility, transfer skills)
- laboratory tests and nutrition
- assessment of psychological health, behavior, and cognitive status
- information on social and financial resources and the availability and utilization of personal care assistance
- assessment of positioning, posture, and related equipment for bed and wheelchair
- assessment of lifestyle, including use of tobacco, past and present, and alcohol and drug use/abuse.[6]

The second phase of assessment consists of a detailed description of the pressure injury itself and the surrounding tissues, including the following factors[6]:

- Etiology and duration of the pressure injury
- Specific anatomical location, general appearance, and characteristics of the wound base
- Size (length, width, depth, and wound area)
- Stage or severity using current terminology and description
- Exudate
- Odor
- Necrosis
- Undermining
- Sinus tracts
- Infection
- Viable tissue (granulation, epithelialization, muscle or subcutaneous tissue)

- Nonviable tissue (eschar, clean nongranulating slough)
- Wound margins and surrounding tissue

Photographs can be useful in these assessments and in monitoring.

The pressure injury should be monitored with each dressing change and the above factors reviewed at regular intervals.

Practice Point

Patients with darkly pigmented skin are particularly vulnerable to undetected pressure injuries. Although areas of damaged skin appear darker than the surrounding skin, tactile information must be used in addition to visual data when assessing persons with darker skin. The skin may be taut and shiny, indurated, and warm to the touch. Color changes may range from purple to blue. Pressure-damaged dark skin doesn't blanch when compressed.[59]

Treatment

Prevention and treatment are inextricably linked across the continuum of care for the person with a pressure injury.[60] During rehabilitation following an SCI, the patient is exposed to a great deal of information about the major physiological changes that have occurred as well as how to prevent or manage potential secondary complications, such as pressure injuries and urinary tract infections. Unfortunately, much of this information is not absorbed during this early posttraumatic phase, resulting in episodes of potentially life-threatening conditions once the patient returns to his or her home and community. Coupled with nonretention of information is today's significant decrease in length of stay, which makes structured education sessions during hospitalization very limited at best or totally absent.

Nonsurgical Treatment

The treatment of pressure injuries is a complex process, based on a number of patient-related and pressure injury–related factors. Nonsurgical treatment for pressure injuries consists of a number of sequential steps that become the treatment plan for that patient with his or her specific injury. The elements of a comprehensive treatment plan include cleansing, debriding, applying dressings, and assessing the need for (and appropriateness of) new technologies aimed at wound healing. Education, in the form of printed materials or discussions with healthcare professionals, is intended to prevent recurrence in the patient with an SCI. Enhanced, individualized pressure injury prevention and management education are effective in improving pressure injury knowledge during hospitalization for either medical management or the surgical repair of a pressure injury.[61,62] Furthermore, initial individualized preventive interventions combined with structured follow-up within a person's individual everyday life setting may reduce the risk of pressure injuries in persons with SCI.[63] Complex interventions of behavioral and education interventions to prevent pressure injuries in adults with spinal cord injury have not been effective in preventing or reducing the occurrence of pressure injuries.[64] However, an individual's preexisting habits may be important when designing a pressure injury prevention and treatment regimen for persons with spinal cord injury. Strategies that promote or encourage habit change can be used to augment pressure injury prevention techniques.[65]

Initially, creating a physiologic wound environment is essential. Cleansing is accomplished with normal saline, sterile water, pH-balanced wound cleanser, or lukewarm potable tap water. Other cleansing agents (such as diluted sodium hypochlorite ¼ to ½ strength for wounds with heavy bioburden for a limited time) and mechanical wound cleansing techniques are also employed depending on the status of the wound and surrounding tissue. Cleansing is followed by debridement of devitalized tissue and selection of wound care dressing. A number of adjunctive therapies have been identified. However, electrical stimulation is one of the very

few to be supported by the scientific evidence. The treatment plan should be modified if the ulcer shows no evidence of healing within 2 to 4 weeks.[6]

Surgical Treatment

Stage 3 and 4 pressure injuries are frequently treated surgically in patients with SCI. The goals of surgical closure include[6,64,65] preventing protein loss through the wound:

- reducing the risk of progressive osteomyelitis and sepsis
- preventing renal failure
- reducing costly and lengthy hospitalization
- improving hygiene and appearance
- expediting time to healing.

The surgical process includes[66,67] excision of the pressure injury and surrounding scar, underlying bursa, soft tissue calcification, and underlying necrotic or infected bone:

- filling dead space with fascia or muscle flaps
- improving vascularity and distribution of pressure over bony prominences
- resurfacing the area with a large flap so that the suture line is away from areas of direct pressure
- providing a flap that leaves options for future surgeries.

Preoperatively, the rehabilitation and surgical teams coordinate their efforts to control local wound infection, improve and maintain nutrition, regulate the bowels, control spasms and contractures, and address comorbid conditions. Previous pressure injury surgery, smoking, UTI, and heterotopic ossification could affect surgical outcomes.[6]

New surgical techniques to repair pressure injuries have been developed and are being used to improve surgical outcomes. Although these techniques are being evaluated,[68–72] reports of long-term follow-up of the status of the skin and recurrence have been limited. One study by Lee[68] used a wound closure technique and followed patients for 102 days, after which 18 of 21 (86%) wounds in 13 patients remained closed. Sorensen et al.[73] suggested that thorough preoperative debridement, patient compliance, control of comorbidities, professional postoperative support, and sufficient pressure relief are essential if surgical success is to be achieved.

Evidence-Based Practice

Postsurgical care includes keeping the surgical site pressure-free, preventing infection, preventing dehiscence of a flap, using specialty beds to maximize pressure reduction, mobilizing the patient progressively, and providing patient and family education.[6]

Support Surfaces

Support surfaces are devices or systems intended to reduce the interface pressure between a patient and his or her bed or wheelchair.[60] Support surfaces neither prevent nor heal pressure injuries; rather, they are prescribed by a clinician and incorporated into a comprehensive pressure injury prevention and management program. Pressure redistribution products, such as active or reactive mattresses, mattress overlays, or specialty beds, may be used at various times to reduce the patient's risk of developing pressure injuries. Reactive support surfaces are used for individuals who have a stable spine and who are able to reposition themselves enough to avoid weight bearing on all areas at risk for pressure injuries. Active support surfaces and high-air-loss or air-fluidized reactive support surfaces are for individuals who have pressure injuries on multiple turning surfaces and/or are status postflap/skin graft within the past 60 days.[6] Bed positioning devices and techniques must be compatible with the bed type and the individual's health status. Individuals should not be positioned directly on a pressure injury. Rather, various positioning aids, such as pillows and cushions, should be used to reduce pressure on areas vulnerable to tissue breakdown. Although there are differences of opinion regarding the frequency of turning or repositioning a person with a pressure injury, the literature supports the importance of individualizing a turning schedule. There are a great number of pressure redistribution bed support surfaces for individuals with or at risk to pressure injuries. The use of these "specialty beds" is determined by the person's status and the individual facility's experience with these devices. These include air-fluidized and low-air-loss devices.

Materials for wheelchair positioning and pressure injury prevention, such as foams and gels, used alone or in combination, and elements such as air and water, also used alone or in combination, are in use across inpatient and home environments. Wheelchairs and seating systems should be specific to the individual and allow that individual to redistribute pressure sufficiently to prevent pressure injuries. The optimum wheelchair seating system should[6]:

- redistribute pressure
- minimize shear
- provide comfort and stability
- reduce heat and moisture
- enhance functional activity
- be inspected at regular intervals for wear.

Wheelchair cushions and seating systems of various materials and designs are intended to reduce pressure and maximize balance and stability when a patient is in a wheelchair. There is no evidence that wheelchair cushions either prevent or heal pressure injuries. Rather, they are used as one important tool in the clinician's armamentarium of prevention and treatment strategies. Power weight-shifting wheelchairs are often prescribed for individuals who are unable to independently perform an effective pressure relief while sitting. Full-time wheelchair users with or without a history of pressure injuries should limit sitting and maintain an off-loaded position from the seating surface for at least 1 to 2 minutes every 30 minutes.[6] (For further discussion on support surfaces, see Chapter 11, Pressure Redistribution: Seating, Positioning, and Support Surfaces.)

Issues surrounding wheelchair sitting and associated seating and positioning devices have been studied extensively and therapeutic strategies developed to minimize pressure on the skin, especially in anatomical areas overlying bony prominences.[74–76] These efforts have led to major improvements in the technology of seating support and pressure-reducing devices such as wheelchair cushions and in mattresses for the supine patient.[6]

Another area remains as yet uninvestigated, namely, the adverse effects of pressure exerted while sitting on a commode chair during bowel care procedures necessary for persons with SCI. Chronic constipation, difficulty in evacuation, incontinence, and damage to mucosa are all complications associated with neurogenic bowel in persons with SCI. One attempt to deal with the adverse effects of pressure during bowel management programs is the padded commode seat. This has led to some success in safety and in reducing the risk of pressure injuries during the bowel management procedures.[77–81] Rates of rehospitalization following SCI remain high

and result from complications in the genitourinary and respiratory systems and pressure injuries.[15] Unfortunately, very little data exist on the specific nature of pressure injury development in association with bowel care sitting time or process. Research is needed that would focus on duration of sitting time associated with the therapeutic approaches to bowel care in persons with spinal cord injury.

Nontraditional Interventions

A number of adjunctive therapies have been reported in the literature with varying degrees of success in treating pressure injuries in the patient with an SCI, including:

- electrical stimulation
- ultraviolet and laser therapy
- hyperbaric oxygen and ultrasound
- negative pressure wound therapy
- nonantibiotic drugs
- topical agents
- skin equivalents
- growth factors.

Among these, only electrical stimulation has enough reported scientific evidence to justify its use as a treatment for pressure injuries in the patient with an SCI.[80–83]

Summary

Although SCI research has improved and expanded tremendously in recent years, designing and conducting randomized, controlled trials that are capable of producing compelling scientific evidence on which to base management of pressure injuries have been disappointing. Advances in pressure injury treatment have included many new products for wound dressings, wheelchair technology, pressure redistribution devices for the bed and wheelchair, and surgical techniques. However, research in pressure injury prevention remains limited. Randomized, controlled trials in real-world settings are the gold standard for assessing the effectiveness of prevention and treatment strategies. However, in complex, rapidly changing healthcare settings, blinding often is not feasible, and it may be impractical to control for every variable that influences a study's outcome. Furthermore, any assumptions that usual care is static are probably mistaken. Innovative approaches to maintain the integrity of the study design must be used, including flexibility in inclusion and exclusion criteria to support accrual, obtaining a better understanding of the important aspects of usual care that may need to be standardized, continuous improvement within the intervention arm, and anticipating and minimizing risks from organizational changes.[31] Research efforts should include prospective studies that focus on preventing recurrence, specifically long-term follow-up programs that promote self-management.

PATIENT SCENARIO

Clinical Data

Mr. K is a 48-year-old African American man who sustained a complete SCI at the T8 level from a motor vehicle crash 7 years ago that resulted in paraplegia. He lives independently, is unmarried and unemployed, and has a history of hypertension. During his rehabilitation hospitalization, Mr. K was told about pressure injuries

and what he could do to prevent them. However, within 1 year after discharge, he developed a stage 3 right ischial pressure injury for which he was hospitalized for surgical repair. The surgery resulted in a healed wound and Mr. K was discharged to his home. Although pressure injury prevention was reviewed during this hospitalization and consumer-based written material was provided, he returned home and resumed previous habits, including sitting in front of the television for long hours, driving around with friends, not eating well, and smoking. Less than a year after the first surgery, Mr. K developed a stage 4 pressure injury with osteomyelitis on his left ischium. He was readmitted to the hospital for the surgical repair of this injury. Compounding Mr. K's pressure injury history are the following: he does not have a social or family support system to help and encourage him to be more proactive in preventing pressure injuries; he seems depressed and unable to take control of his physical health; he would like to return to work as a computer programmer but has not put much effort into looking for a job; he often does not take his prescribed medication for hypertension; he developed type 2 diabetes; and he smokes and sometimes uses alcohol and illegal drugs. His bowel care requires several hours on the commode, contributing to pressure injury recurrence on fragile skin.

Case Discussion

The first priority is to treat the infection (osteomyelitis) and surgically repair the pressure injury. During this hospitalization, a case manager was assigned to coordinate discharge and follow-up plans. The case manager requested that an occupational therapist knowledgeable about SCIs and pressure injuries work with Mr. K to develop strategies to prevent recurrence, help him to take control of the things he can control, identify appropriate support systems within the community and the healthcare system, and improve his quality of life. The occupational therapist worked with Mr. K to develop a pressure injury prevention plan that was acceptable and appropriate to Mr. K's lifestyle. Relevant actions and behaviors were included in a written plan so Mr. K could refer to it daily. These included weight shifts and turns, skin checks (with special attention to the changes in

his darkly pigmented skin), nutrition, hygiene, limiting sitting in one place for long periods of time, consistency with prescription medications, limiting smoking and drinking, and not using drugs. Additionally, the occupational therapist performed a complete assessment of Mr. K's support surfaces used in the bed and wheelchair as well as the wheelchair itself. His wheelchair and wheelchair cushion were in very poor condition. A new wheelchair was ordered and a pressure evaluation performed to identify the most effective wheelchair cushion for him. A mattress overlay, compatible with his bed at home, was also prescribed.

Mr. K was instructed on the routine care of his new equipment (wheelchair, mattress overlay, and cushion), especially with regard to changes in his skin that might reflect the deterioration of the support surfaces. He was re-evaluated for management of his hypertension, and a new medication regimen was initiated. A program to manage his diabetes also was implemented. His bowel regimen was reviewed and modified, and a new commode/shower chair was ordered. A home visit by the occupational therapist identified ways to minimally adapt Mr. K's home environment to maximize his independence and safety. He was referred to a psychologist for counseling that seemed to result in better insight into his behaviors and lifestyle. Finally, Mr. K was given a list of resources he could contact with problems, including the case manager, equipment vendor, physician, and clinics. He also was given information on how to seek employment.

Show What You Know

1. **All of the following are major risk factors for pressure injury development in the SCI population except:**
    A. preexisting conditions.
    B. severity of the SCI.
    C. gender.
    D. nutrition.

2. **The most commonly occurring complications of SCI are:**
    A. fracture.
    B. urinary tract infection.
    C. pressure ulcer development.
    D. pulmonary/respiratory complications.

3. **A pressure injury prevention plan for a person with SCI should always include the following:**
    A. Every 2-hour turning and repositioning schedule
    B. Bowel management regimen with padded commode seat
    C. Off-loading from the wheelchair every 30 minutes for at least 1 to 2 minutes
    D. B and C only

Answers to these questions are found on page 688.

References

1. Weaver, F.M. Spinal Cord Injury QUERI Center Strategic Plan. Hines, IL: Edward Hines Jr. VA Hospital, December 2009. Available at: http://www.queri.research.va.gov/about/strategic_plans/sci-strategic-plan.pdf. Accessed July 20, 2014.
2. National Spinal Cord Injury Statistical Center. “Spinal Cord Injury: Facts and Figures at a Glance.” Journal of Spinal Cord Medicine 37(4):479-80, 2014.
3. Ragnarsson, K.T., et al. “Management of Pulmonary, Cardiovascular, and Metabolic Conditions after Spinal Cord Injury.” In S.L. Stover, et al., eds. Spinal Cord Injury: Clinical Outcomes from the Model Systems. Gaithersburg, MD: Aspen Publishers, Inc., 1995.
4. McKinley, W.O., et al. “Long-term Medical Complications after Traumatic Spinal Cord Injury: A Regional Model Systems Analysis.” Archives of Physical Medicine and Rehabilitation 80(11):1402-10, November 1999.
5. Langemo, D.K., et al. “The Lived Experience of Having a Pressure Ulcer: A Qualitative Analysis.” Advances in Skin & Wound Care 13(5):225-35, September-October 2000.
6. Garber, S.L., et al. Pressure Ulcer Prevention and Treatment Following Spinal Cord Injury: A Clinical Practice Guideline for Health-care Professionals, Consortium for Spinal Cord Medicine Clinical Practice Guidelines. Washington, DC: Paralyzed Veterans of America, 2014.
7. Fuhrer, M.J., Garber, S.L., et al. “Pressure Ulcers in Community-Resident Persons with SCI: Prevalence and Risk factors. Archives of Physical Medicine and Rehabilitation 74(11):1172-7, 1993.
8. Charlifue, S., Lammertise, D.P., Adkins, R.H. “Aging with Spinal Cord Injury: Changes in Selected Health Indices and Life Satisfaction.” Archives of Physical Medicine and Rehabilitation 85:1848-53, 2004.
9. McKinley, W.O., Jackson, A.B., Cardenas, D.D., et al. “Long-term Medical Complications After Traumatic SCI: A Regional Model Systems Analysis.” Archives of Physical Medicine and Rehabilitation 80:1402-10, 1999.
10. Hitzig, S.L., Tonack, M., Campbell, K.A., et al. “Secondary Health Complications in an Aging Canadian SCI Sample.” American Journal of Physical Medicine and Rehabilitation 87:545, 2008.
11. Brienza, D., Krishnan, S., Karg, P., et al. “Predictors of Pressure Ulcer Incidence Following Traumatic Spinal Cord Injury: A Secondary Analysis of a Prospective Longitudinal Study.” Spinal Cord 56(1):28-34, 2017.
12. Smith, B.M., Guihan, M., LaVela, S.L., et al. “Factors Predicting Pressure Ulcers in Veterans with Spinal Cord Injuries.” American Journal of Physical Medicine and Rehabilitation 87(9):750-7, September 2008.
13. Yarkony, G.M., Heinemann, A.W. “Pressure Ulcers.” In S.L. Stover, et al., eds. Spinal Cord Injury: Clinical Outcomes from the Model Systems.

Gaithersburg, MD: Aspen Publishers, 1995.
14. Carlson, C.E., et al. “Incidence and Correlates of Pressure Ulcer Development after Spinal Cord Injury.” Journal of Rehabilitation Nursing Research 1(1):34-40, 1992.
15. Cardenas, D.D., Hoffman, J.M., Kirshblum, S., et al. “Etiology and Incidence of Rehospitalization after Traumatic Spinal Cord Injury: A Multicenter Analysis.” Archives of Physical Medicine and Rehabilitation 85:1757-63, 2004.
16. Niazi, Z.B., et al. “Recurrence of Initial Pressure Ulcer in Persons with Spinal Cord Injuries.” Advances in Wound Care 10(3):38-42, May-June 1997.
17. Goldstein, B. “Neurogenic Skin and Pressure Ulcers.” In M.C. Hammond, ed. Medical Care of Persons with Spinal Cord Injury. Washington, DC: DVA Employee Education System and Government Printing Office, 1998.
18. Salzberg, C.A., et al. “Predicting and Preventing Pressure Ulcers in Adults with Paralysis.” Advances in Wound Care 11(5):237-46, September 1998.
19. Holmes, S.A., et al. “Prevention of Recurrent Pressure Ulcers after Myocutaneous Flap.” Journal of Spinal Cord Medicine 25:S23, 2002.
20. Schryvers, O.I., et al. “Surgical Treatment of Pressure Ulcers: A 20-year Experience.” Archives of Physical Medicine and Rehabilitation 81(12):1556-62, December 2000.
21. Goodman, C.M., et al. “Evaluation of Results and Treatment Variables for Pressure Ulcers in 48 Veteran Spinal Cord-injured Patients.” Annals of Plastic Surgery 43(6):572-4, June 1999.
22. Krause, J.S., Broderick, L. “Patterns of Recurrent Pressure Ulcers after Spinal Cord Injury: Identification of Risk and Protective Factors 5 or More Years after Onset.” Archives of Physical Medicine and Rehabilitation 85:1257-64, 2004.
23. Guihan, M., Garber, S.L., Bombardier, C.H., et al “Predictors of Pressure Ulcer Recurrence in Veterans with Spinal Cord Injury.” Journal of Spinal Cord Medicine 31(5):551-9, 2008.
24. Disa, J.J., et al. “Efficacy of Operative Cure in Pressure Sore Patients.” Plastic and Reconstructive Surgery 89(2):272-8, February 1992.
25. Stal, S., Serure, A., Donovan, W., et al. “The Perioperative Management of the Patient with Pressure Ulcers.” Annals of Plastic Surgery 11:347-56, 1983.
26. Bauer, J., Phillips, L.G. “Pressure Sores.” Plastic and Reconstructive Surgery 121(1 Suppl):1-10, 2008.
27. Tchanque-Fossuo, C.N., Kuzon, W.M. “An Evidence-Based Approach to Pressure Sores.” Plastic and Reconstructive Surgery 127(2):932-9, 2011.
28. Relander, M., Palmer, B. “Recurrence of Surgically Treated Pressure Sores.” Scandinavian Journal of Plastic and Reconstructive Surgery and Hand Surgery 2(1):89-92, 1988.
29. Mandrekas, A.D., Mastorakos, D.P. “Management of Decubitus Ulcers by Musculocutaneous Flaps: A Five-year Experience.” Annals of Plastic Surgery

28(2): 167-74, February 1992.
30. Rubayi, S., et al. “Proximal Femoral Resection and Myocutaneous Flap for Treatment of Pressure Ulcers in Spinal Cord Injury Patients.” Annals of Plastic Surgery 27(2):132-7, August 1991.
31. Guihan, M., Garber, S.L., Bombardier, C.H., et al. “Lessons Learned While Conducting Research on Prevention of Pressure Ulcers in Veterans with Spinal Cord Injury.” Archives of Physical Medicine and Rehabilitation 88:858-61, 2007.
32. Bauer, K., Rock, K., Nazzal, M., et al “Pressure Ulcers in the United States’ Inpatient Population from 2008 to 2012: Results of a Retrospective Nationwide Study.” Ostomy/Wound Management 62(11):30-8, 2016.
33. Leaf Healthcare. The Financial Impact of Pressure Ulcers. A White Paper. 2016. Available at: http://www.leafhealthcare.com/pdfs/LH_WP_FinancialOverview_1563AB_101316.pdf. Last accessed 12 February 2019.
34. Allman, R.M., Goode, P.S., Burst, N., et al. “Pressure Ulcers, Hospital Complications and Disease Severity: Impact on Hospital Costs and Length of Stay.” Advances in Wound Care 12(1):22-30, 1999.
35. Kerstein, M.D. “Unexpected Economics of Ulcer Care Protocols.” Southern Medical Journal 97:135-6, 2004.
36. Garber, S.L., Rintala, D.H. “Pressure Ulcers in Veterans with Spinal Cord Injury: A Retrospective Study.” Journal of Rehabilitation Research and Development 40:433-42, 2003.
37. Javitz, H.S., Ward, M.M., Martens, L. “Major Costs Associated with Pressure Sores.” Journal of Wound Care 7:286-90, 1998.
38. Chan, B.C., Nanwa, N., Mittmann, N., et al. “The Average Cost of Pressure Ulcer Management in a Community Dwelling Spinal Cord Injury Population.” International Wound Journal 10(4):431-40, 2013.
39. Stroupe, K.T., Manheim, L., Evans, C.T., et al. “Cost of Treating Pressure Ulcers for Veterans with Spinal Cord Injury.” Topics in Spinal Cord Injury Rehabilitation 16(4):62-73, 2011.
40. DeVivo, M.J., Chen, Y., et al. “Costs of Care Following SCI.” Topics in Spinal Cord Injury Rehabilitation 16:1-9, 2011.
41. Byrne, D.W., Salzberg, C.A. “Major Risk Factors for Pressure Ulcers in the Spinal Cord Disabled: A Literature Review.” Spinal Cord 34(5):255-63, May 1996.
42. Rintala, D.H. “Quality-of-Life Considerations.” Advances in Wound Care 8(4):71-83, July-August 1995.
43. Chen, Y., DeVivo, M.J., Jackson, A.B. “Pressure Ulcer Prevalence in People with Spinal Cord Injury: Age Period Duration Effects.” Archives of Physical Medicine and Rehabilitation 86:1208-13, 2005.
44. Krause, J.S., Vines, C.L., Farley, T.L., et al. “An Exploratory Study of Pressure Ulcers after Spinal Cord Injury: Relationship to Protective Behaviors

and Risk Factors." Archives of Physical Medicine and Rehabilitation 82:107-13, 2001.
45. Anderson, T.P., Andberg, M.M. "Psychosocial Factors Associated with Pressure Sores." Archives of Physical Medicine and Rehabilitation 60(8):341-6, August 1979.
46. Gordon, W.A., et al. "The Relationship between Pressure Sores and Psychosocial Adjustment in Persons with Spinal Cord Injury." Rehabilitation Psychology 27:185-91, 1982.
47. Krause, J.S., Kjorsvig, J.M. "Mortality after Spinal Cord Injury: A Four-Year Prospective Study." Archives of Physical Medicine and Rehabilitation 73(6):558-63, 1992.
48. Richards, J.S., Osuna, F.J., Jaworski, T.M., et al. "The Effectiveness of Different Methods of Defining Traumatic Brain Injury in Predicting Postdischarge Adjustment in a Spinal Cord Injury Population." Archives of Physical Medicine and Rehabilitation 72(5):275-9, 1991.
49. Pyatak, E.A., Blanche, E.I., Garber, S.L., et al. "Conducting Intervention Research among Underserved Populations: Lessons Learned and Recommendations for Researchers." Archives of Physical Medicine and Rehabilitation 94(6):1190-8, 2013.
50. Clark, F., Rubayi, S., Jackson, J., et al. "The Role of Daily Activities in Pressure Ulcer Development." Advances in Skin & Wound Care 14:52, 54, 2001.
51. Clark, F.A., Jackson, J.M., Scott, M.D., et al. "Data-based Models of How Pressure Ulcers Develop in Daily-Living Contexts of Adults with SCI." Archives of Physical Medicine and Rehabilitation 87:1516-25, 2006.
52. Jackson, J., Carlson, M., Rubayi, S., et al. "Qualitative Study of Principles Pertaining to Lifestyle and Pressure Ulcer Risk in Adults with SCI." Disability and Rehabilitation 32(7):567-78, 2010.
53. Groah, S.L., Schladen, M., Pineda, C.G., et al "Prevention of Pressure Ulcers among People with Spinal Cord Injury: A Systematic Review." PM & R: The Journal of Injury, Function, and Rehabilitation 7(6):613-36, 2015.
54. Garber, S.L., et al. "A Structured Educational Model to Improve Pressure Ulcer Prevention Knowledge in Veterans with Spinal Cord Dysfunction." Journal of Rehabilitation Research and Development 39(5):575-88, September-October 2002.
55. Andberg, M.M., et al. "Improving Skin Care through Patient and Family Training." Topics in Clinical Nursing 5(2):45-54, July 1983.
56. Krouskop, T.A., et al. "The Effectiveness of Preventive Management in Reducing the Occurrence of Pressure Sores." Journal of Rehabilitation Research and Development 20(1):7483, July 1983.
57. Sleight, A.G., Cogan, A.M., Hill, V.A., et al. "Factors Protecting against Pressure Injuries in Medically Underserved Adults with Spinal Cord Injury: A

Qualitative Study." Topics in Spinal Cord Injury Rehabilitation 25(1):31-40, 2019.
58. Schubart, J.R., Hilgart, M., Lyder, C. "Pressure Ulcer Prevention and Management in Spinal Cord-injured Adults: An Analysis of Educational Needs." Advances in Skin & Wound Care 21:322-9, 2008.
59. Bennett, M.A. "Report of the Task Force on the Implications for Darkly Pigmented Intact Skin in the Prediction and Prevention of Pressure Ulcers." Advances in Wound Care 8(6):34-5, November-December 1995.
60. Bergstrom, N., et al. Treatment of Pressure Ulcers, Guideline Report No. 15. Rockville, MD: US Department of Health and Human Services, Public Health Service, Agency for Health Care Policy and Research. AHCPR Publication No. 96-N014, December 1994.
61. Garber, S.L., Rintala, D.H., Holmes, S.A., et al. "A Structured Educational Model to Improve Pressure Ulcer Prevention Knowledge in Veterans with Spinal Cord Dysfunction." Journal of Rehabilitation Research and Development 39:575-88, 2002.
62. Rintala, D.H., Garber, S.L., Friedman, J.D., et al. "Preventing Recurrent Pressure Ulcers in Veterans with Spinal Cord Injury: Impact of a Structured Education and Follow-up Intervention." Archives of Physical Medicine and Rehabilitation 89:1429-41, 2008.
63. Dunn, C.A., Carlson, M., Jackson, J.M., et al. "Response Factors Surrounding Progression of Pressure Ulcers in Community-residing Adults with Spinal Cord Injury." American Journal of Occupational Therapy 63:301-9, 2009.
64. Cogan, A.M., Blanchard, J., Garber, S.L., et al. "Systematic Review of Behavioral and Educational Interventions to Prevent Pressure Ulcers in Adults with Spinal Cord Injury." Clinical Rehabilitation 31(7):871-80, 2017.
65. Fogelberg, D.J., Powell, J.M., Clark, F.A. "The Role of Habit in Recurrent Pressure Ulcers Following Spinal Cord Injury." Scandinavian Journal of Occupational Therapy 23(6):467-76, 2016.
66. Netscher, D., et al. "Surgical Repair of Pressure Ulcers." Plastic Surgery Nursing 16:225-33, 239, Winter 1996.
67. Clamon, J., Netscher, D.T. "General Principles of Flap Reconstruction: Goals for Aesthetic and Functional Outcome." Plastic Surgical Nursing 14:9-14, Spring 1994.
68. Lee, E.T. "A New Wound Closure Achieving and Maintaining Device Using Serial Tightening of Loop Suture and Its Clinical Applications in 15 Consecutive Patients for Up to 102 Days." Annals of Plastic Surgery 53(5):436-41, 2004.
69. Akyurek, M., et al. "A New Flap Design: Neural-island Flap." Plastic and Reconstructive Surgery 114(6): 1467-77, 2004.
70. Ichioka, S., et al. "Triple Coverage of Ischial Ulcers with Adipofascial Turnover and Fasciocutaneous Flaps." Plastic and Reconstructive Surgery

14(4):901-5, 2004.
71. Ichioka, S., et al. “Regenerative Surgery for Sacral Pressure Ulcers Using Collagen Matrix Substitute Dermis (Artificial Dermis).” Annals of Plastic Surgery 54(4):383-9, 2003.
72. Lin, M.T., et al. “Tensor Fasciae Latae Combined with Tangentially Split Vastus Lateralis Musculocutaneous Flap for the Reconstruction of Pressure Sores.” Annals of Plastic Surgery 53(4):343-7, October 2004.
73. Sorensen, J.L., et al. “Surgical Treatment of Pressure Ulcers.” American Journal of Surgery 188(Suppl 1A):42-51, 2004.
74. Garber, S.L. “Wheelchair Cushions: A Historical Review.” American Journal of Occupational Therapy 39:453-9, 1985.
75. Garber, S.L. “Wheelchair Cushions for Spinal Cord-injured Individuals.” American Journal of Occupational Therapy 39:722-5, 1985.
76. Garber, S.L., Dyerly, L.R. “Wheelchair Cushions for Persons with Spinal Cord Injury: An Update.” American Journal of Occupational Therapy 45:550-4, 1991.
77. Malassigne, P., Nelson, A., et al. “Toward the Design of a New Bowel Care Chair for the Spinal Cord Injured: A Pilot Study.” SCI Nursing 10:84-90, 1993.
78. Malassigne, P., Nelson, A.L., et al. “Design of the Advanced Commode-shower Chair for Spinal Cord-injured Individuals.” Journal of Rehabilitation Research and Development 37:373-82, 2000.
79. Nelson, A., Malassigne, P., et al. “Promoting Safe Use of Equipment for Neurogenic Bowel Management.” SCI Nursing 17:119-24, 2000.
80. Nelson, A.L., Malassigne, P., Murray, J. “Comparison of Seat Pressures on Three Bowel Care/Shower Chairs in Spinal Cord Injury.” SCI Nursing 11:105-7, 1994.
81. Nelson, A., Malassigne, P., et al. “Descriptive Study of Bowel Care Practices and Equipment in Spinal Cord Injury.” SCI Nursing 10:65-7, 1993.
82. Baker, L., et al. “Effect of Electrical Stimulation Waveform on Healing of Ulcers in Human Beings with Spinal Cord Injury.” Wound Repair and Regeneration 4(1):21-8, January-February 1996.
83. Wood, J.M., et al. “A Multicenter Study on the Use of Pulsed Low-intensity Direct Current for Healing Chronic Stage II and III Decubitus Ulcers.” Archives of Dermatology 129(8):999-1009, August 1993.

24

Adiposity and the Bariatric Patient

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Objectives

After completing this chapter, you'll be able to:

- discuss healthcare facility concerns to provide care that addresses the needs of the bariatric/obese patient
- identify several skin problems commonly found in the bariatric patient
- discuss unique risk factors and accompanying pressure injury prevention strategies for the bariatric patient.

Introduction

The bariatric population has increased dramatically. Across care settings, healthcare professionals will be caring for persons with higher body mass index (BMI). Therefore, proactive planning, preparation, proper equipment, interdisciplinary teamwork, training, and sensitivity are important hallmarks in providing appropriate care to patients whose weight otherwise interferes with care.

An Important Healthcare Concern

The number of larger, heavier individuals in the United States globally continues to increase, as does the need to provide reasonable accommodation for the bariatric patient population across healthcare practice settings.[1] Demographic studies suggest that more than two-third of all individuals living in the United States are overweight or obese, and 3% to 10% (at least eight million) are morbidly obese, many of which are children.[2] Morbid obesity has essentially quadrupled since the 1980s.[3] Research also shows that the heaviest Americans have become even heavier in the past decade.[4] Studies suggest a substantial increase in obesity among all age, racial, ethnic, and socioeconomic groups.[5] Worldwide, the number of individuals who are overweight or obese, which totals nearly two billion, now exceeds the number of those suffering from starvation.[6,7] These trends combined with the demands of contemporary patient care standards point to an urgent and increasing need for healthcare facilities to address the unique challenges facing the bariatric patient population.

Bariatrics is a general term derived from the Greek word *baros* and is used to refer to the practice of health care relating to the treatment of obesity and associated conditions.[8] The American Society for Metabolic and Bariatric Surgery (ASMBS) defines obesity as a lifelong, progressive, life-threatening, genetically related multifactorial disease of excess fat storage with multiple comorbidities.[9] In contrast to this clinical definition, the National Association to Advance Fat Acceptance (NAAFA) does not consider a person with a high degree of adiposity obese. The association explains that this disapproving term medicalizes a very natural condition. NAAFA reminds us that some people are born tall, some short, some skinny, and ultimately some fat, noting that “We are tired of being labeled in a negative light simply for our God-given habitus.”[10] Regardless of the larger social implications of body types, obesity, according to the National Institutes of Health (NIH), is simply a diagnostic category that represents a complex and multifactorial

condition.[11] These parallel contrasts of terms are vital to understanding the compassionate delivery of outcome-focused patient care as it relates to the bariatric patient. We as care providers do our best work when we adapt a care plan with an acceptance of the needs of the individual patient, and working with the bariatric patient population is no exception.

Hospitals and other healthcare organizations best position themselves to serve patients when size-sensitive policies, equipment, training, and support are in place.[12] This level of preplanning for care is designed to better manage the risk associated with the common, predictable, and preventable consequences of caring for this complex patient. In our discussion of these size-aware practices, we often find it useful to draw the analogy to the established and well-developed field of pediatrics, where patients of widely ranging sizes are deftly accommodated. This tactic can be useful in creating an objective baseline for conversations between care providers as a care plan is developed for an individual in the context of the hospital resources available for meeting that patient's needs.

Classifying the Bariatric Patient

There are a number of ways to measure and define the condition of obesity for purposes of comparing and communicating the scope of a patient's needs among care planners or relating an individual patient to past experiences in order to develop solutions to unique challenges. In the healthcare setting, recognized methods include the following: body weight, BMI, body weight maldistribution, weight-related immobility, or some other factor that interferes with the worker's ability to provide care. BMI is a mathematical formula that assigns relative risk for morbidity and mortality and is often used in healthcare settings to determine the need for special accommodation simply because of this relative risk. BMI is a valuable tool in quantitatively evaluating a patient, but as we will see later, the qualitative characteristics of the individual must be considered as well. The National Heart, Lung, and Blood Institute (NHLBI) provides the following classifications for obesity based on BMI, which is calculated as body weight in kilograms/body height in meters squared.[13,14]

- Overweight: BMI 25 to 29.9
- Class I obesity: BMI 30.0 to 34.9
- Class II obesity: BMI 35.0 to 39.9
- Class III (extreme) obesity: BMI over 40

Moving on to our qualitative descriptions of the obese patient, body weight maldistribution can be a factor in skin assessment, mobility, and risk for pressure injury development or other skin impairment. Bariatric patients are at elevated risk of developing pressure injury due to their body weight and unique distribution.

The waist-to-hip circumference ratio is often used to describe these description differences. Individuals who carry their excess weight in the hips and legs are often referred to as having a "pear-shaped" body maldistribution. This condition places the individual at risk for pressure formation over the buttocks area or anywhere soft tissue protrudes into a shape that can hinder uniform pressure distribution in a given body position. The pear-shaped individual has

a lower ratio but may need special chairs and commodes to accommodate a larger hip size.[1]

The patient with an apple-shaped physique has a greater waist-to-hip ratio and is at greater risk for cardiovascular disease. An “apple-shaped” maldistribution can lead to respiratory challenges as this individual carries their weight predominantly over the upper body, and attention must be directed at positioning the patient to relieve their respiratory structures of this weight, not just initially but over the entire period of time between repositioning.[1] It is important to note that a position that may be comfortable initially may become impossible even over a short period of time. Patients may be reluctant to bring this up if they feel they are burdening staff members by requesting additional repositioning. Further, the patient with upper body weight maldistribution may be at risk for developing skin breakdown if a tracheostomy tube is in place, simply because of the excess tissue in and around the submental region (Fig. 24-1). The submental region is a fascial space of the head and neck (sometimes also termed fascial spaces or tissue spaces). It is the space located between the mylohyoid muscle superiorly and the platysma muscle inferiorly, under the chin in the midline.

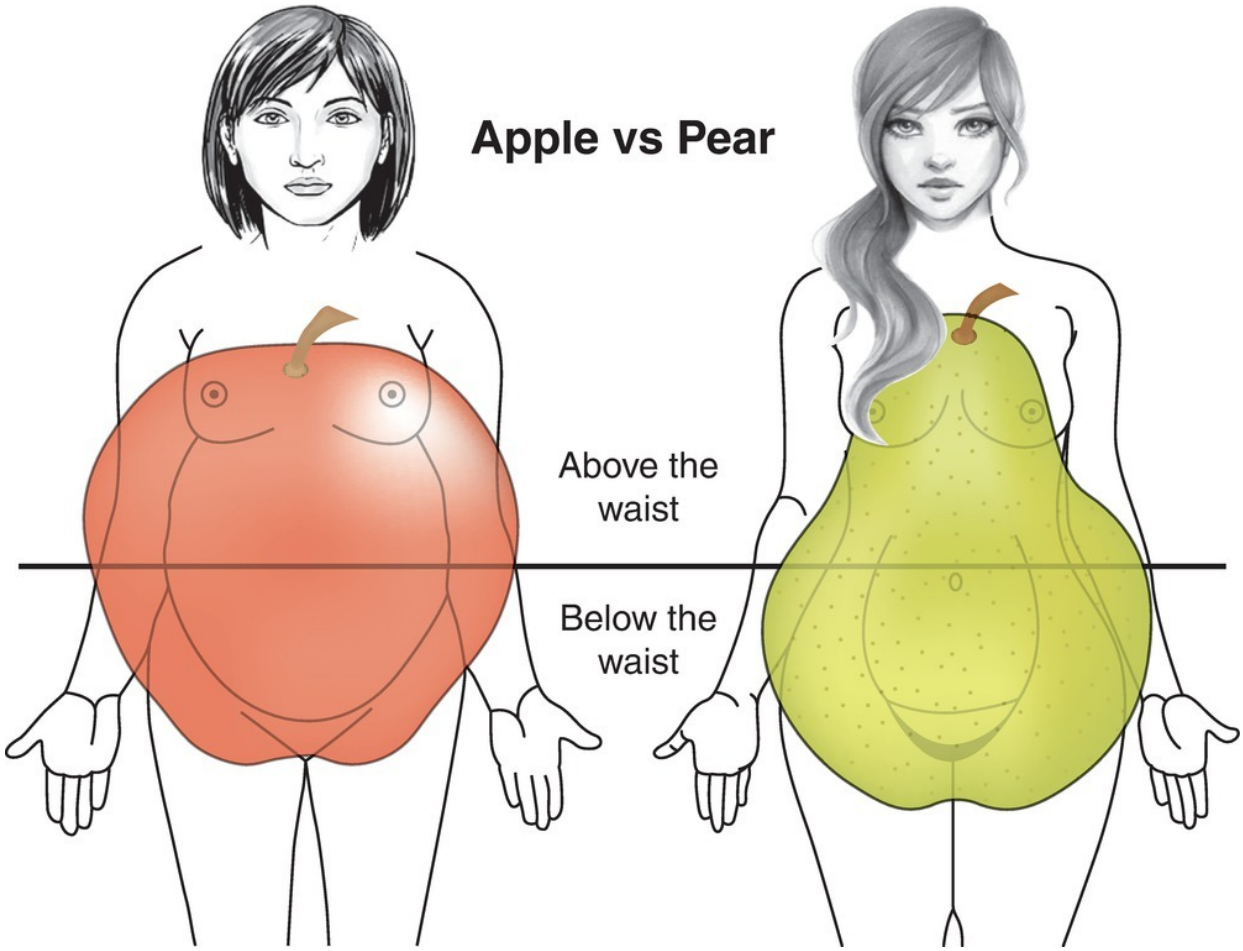


Figure 24-1. Apple versus Pear—Identifying body shapes. The waist-to-hip ratio (WHR) is often used to describe the differences between the two types of body shapes: apple shaped and pear shaped (as shown in these illustrations). The apple-shaped physique is associated with hypertension, cardiac, and respiratory problems. The pear maldistribution places the patient at risk for pressure injury over the buttock region.

Meeting Patient Needs

With ever-increasing obesity rates in the United States, healthcare facilities committed to meeting the needs of this often complex patient population need to have bariatric skin care protocols in place and “ready access” to beds, chairs, commodes, walkers, and other equipment appropriate for bariatric patients. Advanced preparation with regard to protocols and equipment is essential to meet the needs of patients who are obese. Patients frequently present at admission with significant healthcare problems, such as hypertension, type 2 diabetes, coronary artery disease, stroke, gallbladder disease, osteoarthritis, sleep apnea, respiratory problems, an increased risk for certain cancers, and numerous skin manifestations.[1] Despite these risks, bariatric patients may delay medical treatment out of fear or embarrassment, complicating patient care. At this point, the social significance of obesity has the potential to affect clinical outcomes and as such becomes an issue inextricably tied to the clinical treatment of the obese patient.

Practice Point

Ask yourself these questions as you develop your institutional readiness:

- Is your facility prepared to handle the special needs of bariatric patients as they enter the emergency department?
- Are your staff members prepared to provide safe, respectful patient care while avoiding the potential for injury to both patients and staff members?

Many complex considerations are involved in treating bariatric patients, including multiple comorbid conditions; the potential for nonhealing surgical wounds due to dehiscence or infection; a higher risk of venous disease, pressure injury development, and diabetes-related foot wounds; and nutritional concerns. The focus of our discussion is skin assessment and care of the bariatric patient, with the goal of maintaining skin integrity.

Skin Assessment

A complete skin assessment is the first step in any skin care protocol. A cursory assessment of the immediately visible skin is not sufficient. Bariatric patients may have changes in skin physiology due to a greater skin-to-weight ratio, redundant tissue, and poorly perfused adipose tissue that can result in compromised skin health and wound healing. Skin inspection should be performed on admission with the patient wearing a hospital gown and lying flat, if condition tolerates, in bed. All skin folds should be separated gently and examined for any skin changes such as erythematous, moist denuded areas, or pressure injuries. These areas can be extremely painful, so care and assistance (two or more persons) are needed to complete a thorough exam. Common skin fold areas include behind the neck, under the arms and breasts, under the abdomen or panniculus, the flank area, the perineal and rectal area, the upper and lower thighs, and the calf and ankle areas[15] (Fig. 24-2). Many of these issues, as well as the persistence of the pannus, continue in patients following bariatric weight loss surgery, and it is important to identify this category of patient in order to provide them with the same skin integrity assessment upon admission.

Figure 24-2. Skin folds on the back of the neck.

The presence of abdominal adiposity with an accompanying skin fold is formally referred to as a panniculus and more commonly referred to as a pannus (Fig. 24-3). Grading to the extent of being redundant has been helpful in assessment, planning for care, and certain reimbursement issues as they relate to targeted intervention. A scale of grades 1 to 5 was cited in the literature as early as 2000 and has been adopted by a number of groups including the American Society of Plastic Surgery.[15,16]

Figure 24-3. Grade 5 abdominal pannus. (Photo copyright 2006 Coloplast Corp. Used with permission. Photographer: K. L. Kennedy-Evans, RN, CS, FNP.)

- Grade 1: Pannus covers the pubic hairline but not the entire mons pubis.
- Grade 2: Pannus extends to cover the entire mons pubis.
- Grade 3: Pannus extends to cover the upper thigh.
- Grade 4: Pannus extends to midthigh.
- Grade 5: Pannus extends to the knee and below.

Forces such as the pressures between skin folds and moisture accumulation, as an aggravating factor, can lead to deep tissue injury, pressure injury, or intertriginous dermatitis (ITD) (Fig. 24-4). Ongoing skin assessment and appropriate interventions are critical in maintaining and healing any skin manifestations or ulcers. The EPUAP/NPIAP//PPPIA 2019 Pressure Injury/Ulcer Prevention and Treatment Guideline for Bariatric (Obese) Individuals provides assessment and positioning recommendations.[17] (See *Assessment and positioning recommendations for bariatric patients*.)

Figure 24-4. Grade 5 pannus with intertriginous dermatitis. (Photo courtesy of C. Fife, MD.)

When examining the skin, you may find certain benign conditions or more serious infections that commonly occur in obese individuals. These include the following:

- Acanthosis nigricans: Hyperpigmented (often brown) irregular plaques in skin folds (especially the axilla and back of the neck) (Fig. 24-5). The plaques often feel like velvet when touched but may eventually become rougher in texture. This condition is associated with obesity as well as other systemic diseases, such as insulin resistance, diabetes, hormonal changes such as hypothyroidism, cancer, and more. In many situations, treating the underlying problem can help fade the discoloration. Examples may

include weight loss, medication, or surgery to address the underlying cause. If the skin surface becomes aesthetically problematic or uncomfortable for the patient, consider prescription creams to lighten the area, antibacterial soaps to reduce odor, or laser therapy to reduce the thickness of the skin surface.[18]

- Plantar hyperkeratosis: Excessive weight on the feet leads to thickening of the weight-bearing surfaces on the soles (Fig. 24-6). Treatment includes weight loss and orthotic shoe inserts to protect bony prominences from breakdown.
- Stasis Dermatitis: Erythema and scaling seen in the lower extremities due to impaired venous circulation. Commonly seen in obese individuals or secondary to venous thrombosis. Presents with itching, red, and dry areas. The skin may appear shiny or have reddish-brown patches. Swelling (edema) is usually present. In chronic cases, there is gradual tightening and scarring of the skin leading to hard and sometimes tender areas. In severe cases, the skin breaks down with oozing, crusted areas, and ulceration (Fig. 24-7A and B).
- Charcot Foot: About 62% of adults living in the United States with type 2 diabetes are obese, and 21% are morbidly obese. Charcot foot is increasing in prevalence among those individuals with morbid obesity who also have diabetes-related neuropathy. This poses a two-fold challenge: impaired sensation and difficulty visualizing changes in the feet (Fig. 24-8A and B).
- Cellulitis: A common, potentially serious bacterial skin infection. The affected skin appears swollen and red and is typically painful and warm to touch. Cellulitis usually affects the skin on the lower legs, but it can occur on the face, arms, and other areas. It occurs when a crack or break in your skin allows bacteria to enter (Fig. 24-9A and B).

Figure 24-5. Acanthosis nigricans. A patient with brown velvety hyperpigmentation of the skin often seen in over weight patients.

Assessment and Positioning Recommendations for Bariatric Patients[1,17]

- Request adequate staff assistance to fully inspect all skin folds.
- Atypical pressure injuries may develop over the buttocks, buttocks cleft, or other areas because of maldistribution of weight as well as between skin folds and/or in locations where tubes and catheters burrow into soft tissue.
- Check that tubes, catheters, and other devices or objects are not underneath the patient or between skin folds.
- Consider slings and bands used with a ceiling or floor-based lift for patient repositioning, numerous types are

available; consider limb or pannus slings to safely access hard-to-reach areas.
- Use pillows or other positioning devices to off-load pannus or other large skin fold mass and prevent skin-on-skin pressure.

More serious skin problems are often found during initial assessment, such as cellulitis; macules, papules, or pustules (especially to the neck and back); lymphedema; hemosiderosis and other skin changes associated with venous insufficiency of the lower extremities; venous ulcers; dehiscence of surgical wounds; moisture-associated skin damage; and pressure injury.[19–21] Many of these conditions are covered in other chapters of this book. Here, we give special consideration to the prevention and treatment of moisture-associated skin damage and pressure injuries specifically as they relate to bariatric patients.

Moisture-Associated Skin Damage

ITD is one of four types of moisture-associated skin damage and is sometimes referred to as intertrigo. This condition results from moisture trapped within skin folds. ITD may occur in the presence of bacterial, fungal, or viral involvement and is simply inflammation of the skin folds.[22,23] These inflammatory changes often present as mirror images where one skin surface touches another. Although this condition can be found in any patient, obese patients are at greater risk because of the nature of skin folds that accompany an obese habitus. For example, Brown and colleagues[20] surveyed 100 obese patients for the prevalence of skin problems, including rash, dryness, and "broken skin." Of the respondents, 63% indicated that they had more than one skin problem, the most common being broken skin and itching.[20] Aggravating factors that impact the frequency and severity of ITD among the individual include moisture, such as perspiration, urine, feces, mucus, or wound drainage. Moisture from these various sources becomes trapped under or within skin folds creating maceration; pressure from large skin folds on underlying skin creates areas of pressure-induced injury; friction as one skin surface moves across another creates shear with movement resulting in fissures at the base of the skin fold; physical challenges in maintaining hygiene and the warm, dark, moist conditions that favor microbial factors all contribute to the persistence of these issues if not addressed through timely interventions; and/or prevention. Additionally, cellulitis becomes a risk if the condition is not treated in a timely, appropriate manner (Fig. 24-10).[23]

ITD can develop in any skin fold but is most common under the breasts, abdominal skin folds (pannus), and axillae as well as in the submaxillary area and groin or perineum. Obese patients may report a history of skin irritation under or within skin folds. Combining factors, such as bed rest, immobility, fever, and the use of such medications as antibiotics and steroids, often increase the risk of recurrence once the patient is hospitalized.

Preventing Intertriginous Dermatitis

Prevention of ITD is a key component of any bariatric skin care protocol. Preventive interventions focus on keeping the skin clean, dry, and well supported in order to minimize the cumulative effects of moisture, pressure, friction, and shear. Use a gentle ph balanced soap or no-rinse skin cleanser for bathing and follow up by patting skin dry with a soft cloth. Because moisture accumulation between skin folds is an ongoing problem between bathing, it is essential to ensure that the area is clean and dry to begin with. Between bathing, soft absorbent pads, such as soft linen or nonocclusive high airflow incontinence pads, can be placed between skin folds to off-load pressure, absorb moisture, and lessen friction and shear with movement. Some facilities have had success with a silver-impregnated fabric, which is specifically designed to be placed in the skin folds of bariatric patients (Fig. 24-11). A properly fitted nonsynthetic bra may also help achieve these goals in large-breasted women. Some men can also have large breasts and will need support as well. Any material placed between the skin folds should be changed at frequent intervals or whenever it accumulates moisture.

When repositioning patients in bed, make sure that the pannus is well supported with a pillow and separate the legs by placing a pillow between the knees to make sure there is adequate airflow to skin fold areas. Low air loss beds or those addressing microclimate may help dry moist areas.

Treating Intertriginous Dermatitis

A wide variety of products have been used to treat ITD; however, the most effective treatments are based on an analysis of the underlying etiology. *Candida* should be treated with topical antifungal products; systemic antifungal agents such as fluconazole may be necessary in severe cases. Likewise, bacterial infections of the skin may be treated with topical antibiotics or if the condition is not improving (or if it is progressing to cellulitis), systemic antibiotics. Topical and systemic steroids may be useful in cases of atopic or contact dermatitis.[24] Beyond these treatments, consider burrow solution (aluminum acetate) soak applied for 15 to 20 minutes twice per day to soothe and dry affected area.[12]

Unfortunately, ID is especially prevalent among obese patients after weight loss because of the extent of redundant skin. Panniculectomy and other forms of recontouring surgery may be necessary to remove excessive skin folds after weight loss has been achieved.[15]

Figure 24-6. Plantar hyperkeratosis. Hyperkeratosis results from chronic excessive pressure or friction to the epidermis, as seen here on the plantar aspect of the foot.

Figure 24-7. Stasis (Venous) Dermatitis. **(A and B)** A common inflammatory skin disease that occurs on the lower extremities. Bilateral erythema and scaling seen in the lower extremities due to impaired venous circulation.

A

Figure 24-8. (A) Charcot foot. **(B)** Charcot foot. A condition causing weakening of the bones in the foot that can occur in people who have significant nerve damage (neuropathy). The bones are weakened enough to fracture, and with continued walking, the foot eventually changes shape. As the disorder progresses, the joints collapse and the foot takes on an abnormal shape, such as a rocker-bottom appearance. (Photos courtesy of J. Niezgoda.)

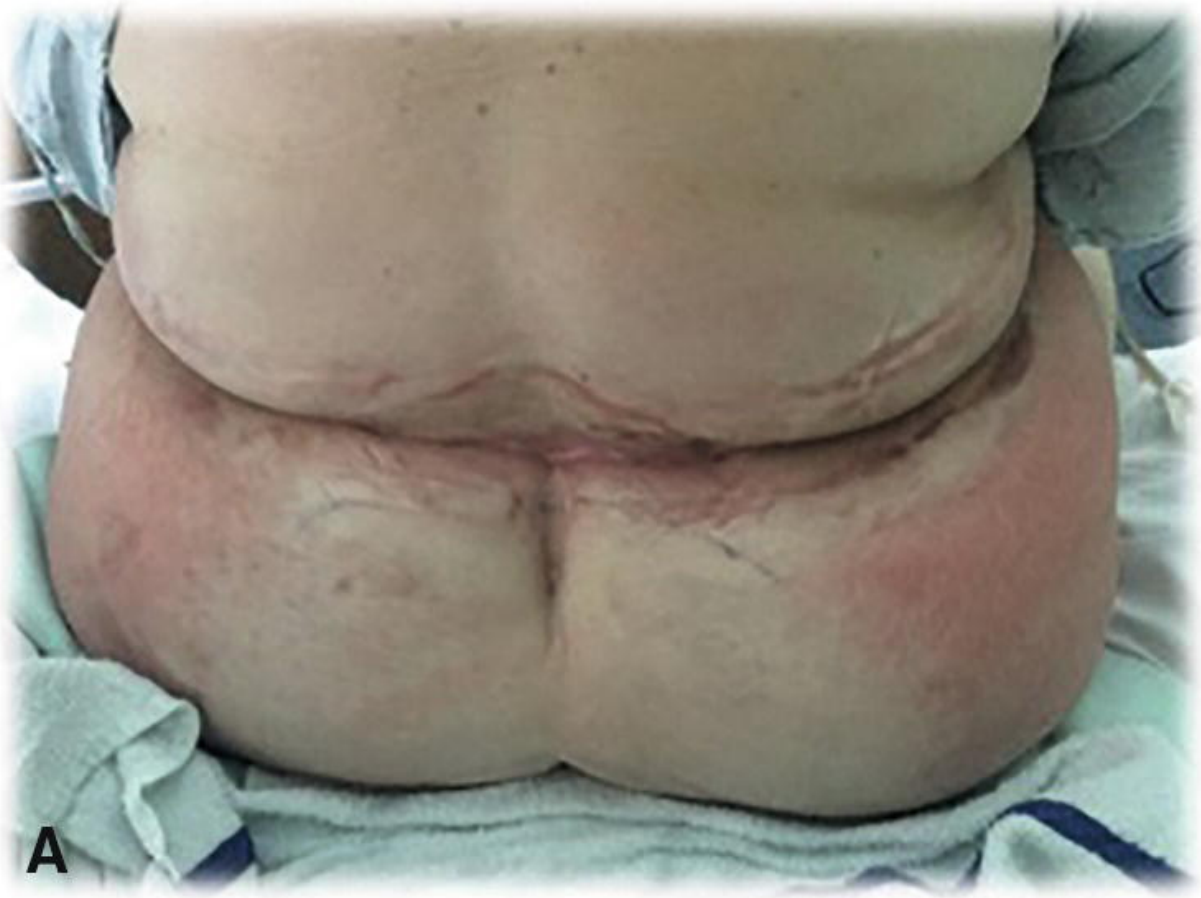

A

Figure 24-9. Bacterial infection involving the inner layers of the skin. Signs and symptoms include an area of redness, which increases in size over a few days. **(A)** Note that the cellulitis has spread into the pannus area in Figure A. **(B)** Leg swollen, red, warm, and tender to touch. (Photos courtesy of Kimberly LeBlanc **[A]** & Jeffrey A. Niezgoda **[B]**)

A

B

Figure 24-10. Intertriginous dermatitis. **(A)** Intertriginous dermatitis between the skin folds of the breast of a female patient with a body mass index of 60. Note the fissure at the base of the skin fold. **(B)** Intertriginous dermatitis with erythema, erosion, and denudation. **(C)** Candida involvement in a skin fold. (Photos courtesy of K. L. Kennedy.)

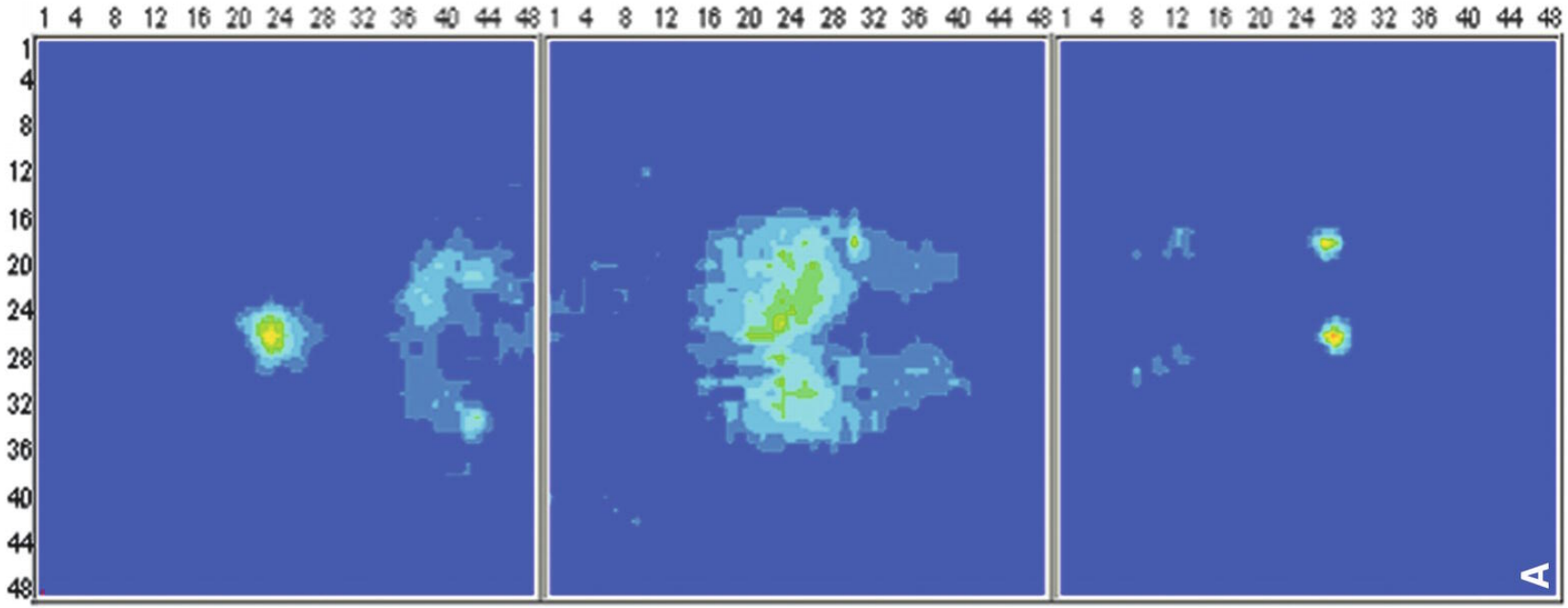
Figure 24-11. InterDry. A product used to prevent and treat intertriginous dermatitis in bariatric patients.

Pressure Injury Prevention

General pressure injury prevention is discussed in Chapter 14; however, the discussion in this chapter focuses on the unique risk factors and needs of the bariatric patient in preventing pressure injuries.

Unique Risk Factors

Several factors increase pressure injury risk in the bariatric patient. Laboratory pressure studies indicate that pressure is distributed somewhat differently in obese patients. Among the general patient population, high-pressure areas in the supine position are predominantly over bony prominences (e.g., the head, sacrum, and heels). The obese patient presents a contrast to these guidelines with a large amount of weight-induced force distributed over the entire supine surface. Pressures may be high over bony prominences, but high-pressure areas are also seen in traditionally soft tissue areas such as the buttocks. Even though the surface area is larger in an obese patient, there is still often greater tissue weight than normal on traditionally soft tissue areas (Fig. 24-12). This is complicated by maldistribution, emphasizing the need for individual assessment of high-pressure areas unique to the patient's body habitus.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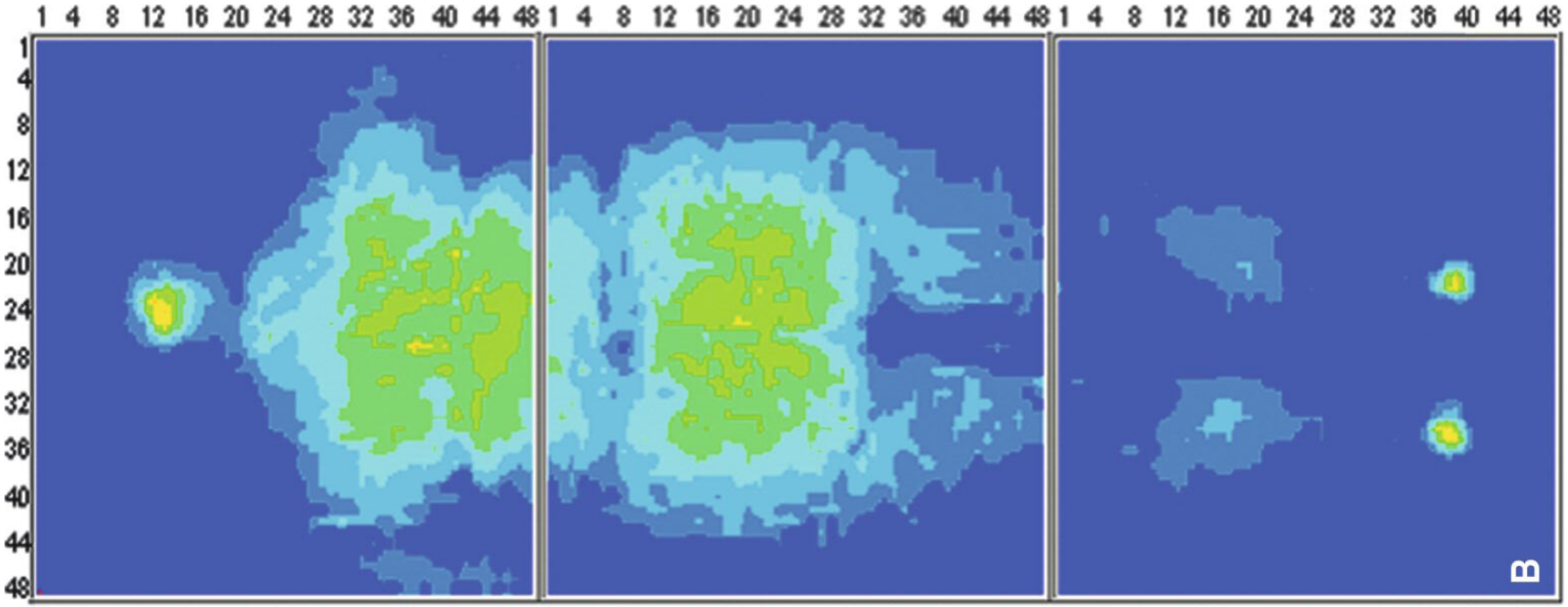
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Figure 24-12. Pressure distribution. **(A)** High-pressure areas are more focused over bony prominences in patients with a lower body mass index (BMI). **(B)** A more diffuse pattern of high-pressure distribution is evident in the patient with a higher BMI. High-pressure areas occur over soft tissue areas as well as bony prominences. Note enlarged yellow area indicating more pressure in the buttock region.

Assessment

Pressure injuries that do develop in obese patients often have slightly different characteristics. They develop not only over bony prominences but also over other areas of high tissue pressure, such as the buttocks (see Fig. 24-12). Bilateral hip pressure injuries are commonly seen in patients placed in chairs that are too narrow for their hip width.[6] Bariatric patients also seem to be at higher risk for pressure damage related to medical devices (such as tubes, oxygen tubing, endotracheal tubes, oximetry probes, and tight narrow tracheostomy ties).[22] These factors demand strategies that address the potential for pressure injuries caused by often innocuous sources and require an elevated awareness of these risks.

Standard risk assessment tools such as the Braden Scale are still important as general screening measures for pressure injury risk status. However, consider exploring each subscale more fully when caring for an obese patient. Many obesity-related pressure injuries can be prevented with proper risk assessment, selection of bariatric-sized equipment technology and devices, proper application of same, and frequent inspection of the skin for areas at risk for pressure-related damage or other tissue damage.[25]

Deep Tissue Injury

There is some clinical speculation that deep tissue injury (DTI) may also be more common in obese patients. Deep pressure may predispose the patient to deep injury. When assessing the skin of bariatric patients, check carefully for subtle signs of DTI, such as slight discoloration of the skin, and changes in skin temperature and texture. If deep tissue injury is suspected, off-load the affected area in an attempt to reduce pressure and salvage any tissue that is injured but not ischemic.[23]

Consider the following questions when planning care for your bariatric/obese patients:

- For patients on bed rest, what positioning strategies will improve respiration without increasing the risk of shear injury?
- Can pillows be placed under the arms to prevent sliding and shear? Would a trapeze over the bed help with patient mobility?
- Is the bed wide enough for the patient to turn comfortably from side to side?
- What is the patient's nutritional status?
- If incontinence-associated dermatitis is a risk, could it be improved by providing a bariatric bedside commode or walker to ambulate to the bathroom? Is a properly sized lift and sling available to transfer the immobile patient? Is a sit-to-stand or other mobility device available to promote safe transfer to a bedside commode?

Bariatric Equipment

Planning care for the bariatric patient population brings many specific concerns to the surface, which need to be considered in order to provide care that is safe and effective in addressing the unique needs of larger, heavier patients. Areas of particular concern include safety (both for the patient and the caregiver), accessibility for the patient, sensitivity of all caregivers and staff members, use of specialized

technology and devices that will work for larger patients, and sufficient staffing.[25]

Ensuring a facility's readiness to provide for patient safety and comfort is essential and can be assessed by performing a survey of the facility, paying specific attention to weight limits of equipment, including chairs, beds, commodes, shower seats, physical therapy tables, lift/transfer equipment, carts, exam tables, wheelchairs, walkers, CT scanner, MRI scanner, OR table, and any other equipment needed to provide care for any patient. All equipment should be labeled in a way making it easier for staff to be aware of weight restrictions, while not publicizing that it is "bariatric equipment." Subtle methods include "EC 500" or "U500" for extended capacity to 500 pounds or under 500 pounds. Check to see if toilets are floor or wall mounted, as wall-mounted toilets may need to be retrofitted to meet weight capacity requirements. Standard wall-mounted toilets can be adapted to accommodate bariatric patients by using a relatively inexpensive support that is available commercially. Additionally, it is necessary to look at the width of doorways to assure larger equipment will fit into and out of the room. This is an important preventative measure because engineering constraints in the moment potentiate patient and caregiver injury. There should be room for a patient to ambulate through the doorway with a larger walker and with a healthcare professional on either side for stability, since this may be what is needed for safety.[4] Additionally, consider the width, length, and weight limits of elevators.[26] The combination of these accommodations can make or break the patient experience for the bariatric patient, beyond the immediate safety implications.

Larger-sized personal items such as gowns, robes, slippers, identification bands, blood pressure cuffs, antiembolus stockings, sequential compression devices, and even gait belts need to be available for patients so that they can be cared for with dignity and safety. Just as we would not put a standard-sized gown on a pediatric patient, it is not acceptable to snap together two standard gowns to fit around an obese patient.[26] Again, the immediate availability of these items improves patient satisfaction as well as patient outcomes.

Bariatric Bed and Chair Considerations

When selecting a bariatric bed frame, some basic criteria should be considered. Ask yourself these questions[26]:

- Is the bed frame wide enough? Does the bed frame have the capacity to expand and reduce the width to meet the patient's clinical care needs?
- Can the patient turn and reposition himself in the bed? For example, confirm that the patient's girth does not touch the side rails of the bed frame when the patient is turned from side to side.
- What is the weight capacity of the bed frame? Does the patient have general obesity or a maldistribution that influences the ability to raise and lower the foot or head feature?
- Is a trapeze (or other supportive structure) available so that the patient can assist with turning? If the trapeze is available, has the device been load tested to accommodate the patient's weight and activity?
- Does the therapeutic support surface have features to assist with moisture control/microclimate if indicated? Consider using features that provide airflow over the surface of the skin to facilitate fluid evaporation if the skin is excessively moist.
- Are bed frames and support surfaces designed for safe and easy patient egress to help facilitate early progressive mobility?
- Are wheelchairs and chairs in the room wide enough to accommodate the individual at his maximum width?

Staff Concerns

The American Nurses Association recently published the Safe Patient Handling and Mobility (SPHM) Interprofessional National Standards[27]; shortly thereafter, the Implementation Guide to the Standards was published.[28] The interest in national and state SPHM legislation is driving worker safety initiatives. The goal of SPHM is to improve patient safety while preventing caregiver injury. It was not

until recently that science helped caregivers and consumers understand the risks of manual handling in the patient care areas.[29] This concern is increasingly significant when caring for the bariatric patient.[30] To that end, worker safety endeavors raise a number of relevant questions with regard to caring for bariatric patient. For example, can staff members transport the patient in the bed? Does the bed fit through the doors throughout the entire hospital? Can the bed be steered easily? Does the bed have a power drive feature? Are bariatric-sized lifts and air transfer devices available? Have staff members been properly trained to use bariatric equipment to ensure both staff and patient safety? Eliminating the fear of injury while providing care requires education of the staff with regard to proper lifting and handling techniques, as well as model-specific training on available equipment. It should be a part of an organization's educational program to fully prepare all patient care staff on the safe handling of the bariatric patient. This will prevent injuries and provide a high level of care that diminishes tissue damage and reduces the inherent risks associated with mobilizing bariatric patients.[31]

Repositioning and Care

Repositioning the bariatric patient is both an art and a science, paying heed to established protocol as well as the needs of the individual patient. Routine turning should be accomplished by a team of trained caregivers in a manner that preserves the patient's dignity. When possible, enlist the patient's help in turning in order to maintain and progress their mobility. Patients and their families often have very useful suggestions for achieving and maintaining mobility despite challenges. These suggestions based on experience are not only helpful to care staff, but they have the potential to better include the patient and family in the larger care plan. The benefits of this collaboration extend postdischarge and have the potential to decrease the likelihood of readmission.[26] The frequency of repositioning should be based on the individual's ability to tolerate turning and repositioning. Bariatric patients who cannot reposition themselves should be assessed frequently and repositioned as often as needed using universally recognized protocols or algorithms.[1]

When repositioning bariatric patients in bed, several strategies are particularly important. Be sure to off-load bony prominences just as we do with the general patient population. Rather than relying on repositioning pillows under the patient's shoulders and hips, the patient should be turned sufficiently to avoid areas of potential pressure. Proper technology (equipment and devices) may be required to prevent friction and shearing injuries when repositioning. Manually grabbing and pulling on a patient may cause skin tears as well as shoulder injuries. This risk must be considered against mobility alternatives that may better suit the individual's skin condition.[32] As far back as 1995, Oertwich and colleagues[33] suggested that small shifts in body weight may be effective in promoting skin health. Careful consideration to the pannus region and considering small shifts may also be helpful. However, small shifts are intended as an adjunct, not a replacement, to regular repositioning in high-risk patients.

Off-Loading Heels

Because of compromised circulation, heels may pose a significant risk to skin health. Heels should be supported off the surface of the bed by placing pillows under the calf and avoid pressure on the tendon or with the use of positioning devices. Examine the unique anatomy of your patient's lower extremities as the heel area may be ambiguous due to maldistribution or edema. Additionally, patients with large calves may be naturally suspending their heels off the surface of the bed, shifting caregiver concern from the pressure on the heel to the pressure distributed on the calf.[17] The unique sizes and shapes of individuals require that care staff feel under the heel and leg to note any high-pressure areas that can be relieved with properly placed pillows to redistribute weight. When using heel positioning booth or devices, make sure they are properly sized for your patient and can be applied without creating high-pressure areas under straps or other areas of the device. The goal of redistribution in an individual's care plan is even distribution of pressure; redistribution that shifts pressure concentration from one area to another over any period of time is not going to be as effective as a subjective analysis

of the patients' unique pressure footprint throughout their positions of rotation.

Summary

The prevalence of obesity continues to climb in the United States and worldwide, resulting in an increasing number of bariatric patients admitted to hospitals each day. As this trend progresses, the international community is looking to the United States for solutions to improving care for the bariatric patient, and American health care is constantly innovating in this field. Caregivers everywhere best serve patients when they understand the unique and complex challenges associated with safe, quality, sensitive patient care. Preplanning for care is the first step to success, along with targeted assessment, policies, and procedures, size-appropriate technology, training, and outcomes measurement.[34] The combination of preparation, training, and the availability of treatment and mobility options is a foundation upon a respectful individualized care plan for the bariatric/obese patient can be tailored. This approach ensures the highest quality care for conditions presented upon admission, as well as interventions aimed at preventing pressure injuries during hospital stays. The results are evident in patient outcomes and worker safety and satisfaction.[26]

Show What You Know

1. **Mirror image inflammation in the skin folds of the obese patient is called:**
   A. a skin tag.
   B. stretch marks.
   C. intertriginous dermatitis.
   D. hyperkeratosis.

2. **Acanthosis nigricans is always a benign condition for which there is no treatment.**
   A. True
   B. False

3. **Proper positioning for the bariatric patient confined to bed includes:**
   A. using small shifts in body weight to replace turning.
   B. keeping the head of the bed flat at all times to prevent shear.
   C. avoiding pillows between the legs.
   D. supporting the pannus and other large skin folds.

***Answers to these questions are found on page 688*.**

References

1. Gallagher, S., Hilton, T., Muir, M. “Safe patient handling & mobility: Bariatric considerations.” American Journal of Safe Patient Handling and Movement 4(2 Suppl):S1-S16, 2014.
2. National Institute of Diabetes and Digestive and Kidney Disorders. “Overweight and Obesity Statistics.” Available at: https://www.niddk.nih.gov/health-information/health-statistics/overweight-obesity. Accessed April 26, 2019.
3. Gallagher, S.M. “The Intersection of Ostomy and Wound Management, Obesity, and Associated Science.” Ostomy/Wound Management 60(1):6-7, 2014.
4. Bolanle, AA. “Waist Circumference Disparity: A Comparison of American, European and Asian Cut Offs Among Africans.” Journal of Applied Biotechnology & Bioengineering 4(3):605-8, 2017.
5. The State of Obesity: Better Policies for a Healthier America. “Socioeconomics and Obesity.” Available at: https://www.stateofobesity.org/socioeconomics-obesity. Accessed April 26, 2019.
6. Heymsfield, S.B., Wadden, T.A. “Mechanisms, Pathophysiology and Management of Obesity.” The New England Journal of Medicine 376:254-66, 2017.
7. World Health Organization. Obesity and Overweight 2013. Available at: http://www.who.int/mediacentre/factsheets/FS311/EN. Accessed April 26, 2019.
8. Camden, S. Obesity: An Emerging Concern for Patients and Nurses. OJIN: The Online Journal of Issues in Nursing. Available at: http://www.nursingworld.org/MainMenuCategories/ANAMarketplace/ANAPeriodicals/OJIN/TableofContents/Vol142009/No1Jan09/Obesity-An-Emerging-Concern.aspx. Accessed April 26, 2019.
9. American Society for Metabolic and Bariatric Surgery. The Disease of Obesity. Available at: http://www.asmbs.org. Accessed April 26, 2019.
10. National Association to Advance Fat Acceptance. Available at: http://www.naafaonline.com/dev2/. Accessed April 26, 2019.
11. Flegal, K.M., Carroll, M.D., Kit, B.K., et al. “Prevalence of Obesity and Trends in the Distribution of Body Mass Index Among US Adults, 1999-2010.” JAMA 307(5):491-7, 2012.
12. Gallagher, S.M. “Skin and Wound Care among Obese Patients.” In T.A. Bryan, D. Nix, eds. Acute and Chronic Wounds. St Louis, MO: Mosby, 2015.
13. National Institutes of Health. BMI Table. Available at: http://www.nhlbi.nih.gov/guidelines/obesity/bmi_ tbl.pdf. Accessed April 26,

2019.
14. National Institutes of Health. Classification of Obesity and Overweight. Available at: http://www.nhlbi.nih.gov/guidelines/obesity/e_txtbk/txgd/414.htm. Accessed April 26, 2019.
15. American Society of Plastic Surgery. Practice Parameter for Surgical Treatment of Skin Redundancy for Obese and Massive Weight Loss Patients. Available at: http://www.plasticsurgery.org/Documents/medical-professionals/health. Accessed April 26, 2019.
16. Igwe, D. Jr, Stanczyk, M., Lee, H. “Panniculectomy Adjuvant to Obesity Surgery.” Obesity Surgery 10:530, 2000.
17. European Pressure Ulcer Advisory Panel, National Pressure Injury Advisory Panel, and Pan Pacific Pressure Ulcer Injury Alliance. In: E. Haesler, ed. Prevention and Treatment of Pressure Ulcers/Injuries: Clinical Practice Guideline. The International Guideline. 2019.
18. Mayo Clinic. Acanthosis Nigricans. Available at: http://www.mayoclinic.org/diseases-conditions/acanthosis-nigricans/basics/treatment/con-20025600. Accessed April 26, 2019.
19. Beitz, J.M. “Providing Quality Skin and Wound Care for the Bariatric Patient: An Overview of Clinical Challenges,” Ostomy/Wound Management 60(1):12-21, 2014.
20. Brown, J., Wimpenny, P., Maughan, H., “Skin Problems in People with Obesity.” Nursing Standard 18:38-42, 2004.
21. Baranoski, S. Skin Conditions Frequently found in the Obese Population. Wound Source Blog. August 10, 2018.
22. McNichol, L.L., Ayello, E.A., et al. “Incontinence-Associated Dermatitis: State of the Science and Knowledge Translation”. Advances in skin & wound care 31(11):1-12, 2018.
23. Black, J.M., Gray, M., Bliss, D.Z., et al. “MASD Part 2: Incontinence-Associated Dermatitis and Intertriginous Dermatitis.” Journal of Wound, Ostomy, and Continence Nursing 38(4):359-70, 2011.
24. Cheung, D.S., Maygers, J., Khouri-Stevens, Z., et al. “Failure Modes and Effects Analysis: Minimizing Harm to our Bariatric Patients.” Bariatric Nursing and Surgical Patient Care 1(2):107-14, 2006.
25. Gallagher, S.M. “Special Patient Populations.” In W. Charney, ed. Epidemic of Medical Errors and Hospital-Acquired Infections. Boca Raton, FL: CRC Press, 2012.
26. United States Department of Veterans Affairs. 2018 Safe Bariatric Patient Handling Toolkit. Available at: http://www.visn8.med.va.gov/patientsafetycenter/safePtHandling/toolkitBariatrics.aspVAToolkit. Accessed April 26, 2019.
27. American Nurses Association. Safe Patient Handling and Mobility Interprofessional National Standards: Implementation Guide. Silver Spring,

MD: ANA Nursing World, 2013.
28. Gallagher, S.M. American Nurses Association Implementation Guide to Safe Patient Handling and Mobility Interprofessional National Standards. Silver Spring, MD: ANA Nursing World, 2013.
29. Marras, W. Back Breaking Work: Science Video. Available at: http://www.sciencentral.com/articles/view.php3?article_id=218392911&cat=3_all. Accessed April 26, 2019.
30. Randall, S.B., Pories, W.J., Pearson, A., et al. “Expanded Occupational Safety and Health Administration 300 Log as Metric for Bariatric Patient-Handling Staff Injuries.” Surgery for Obesity and Related Diseases 5(4):463-8, 2009.
31. Gallagher, S.M. “Exploring the Relationship Between Obesity, Patient Safety, and Caregiver Injury.” American Journal of Safe Patient Handling and Movement 1(2): 8-12, 2011.
32. Gallagher, S.M. Arnold, M. “The Shoulder: Lessons Learned.” American Journal of Safe Patient Handling and Movement 3(3):8-13, 2012.
33. Oertwich, P.A., Kindschuh, A.M., Bergstrom, N. “The Effects of Small Shifts in Body Weight on Blood Flow and Interface Pressure.” Research in Nursing & Health 18(6):481-8, 1995.
34. Gallagher, S.M. “The Meaning of Safety in Caring for the Larger, Heavier Patient.” In W. Charney. Handbook of Modern Hospital Safety. Baca Raton, FL: CRC Press, 2011.

25

Pressure Injuries in Neonatal and Pediatric Populations

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Objectives

After completing this chapter, you will be able to:

- identify pediatric pressure injury risk assessment tools
- describe factors that place neonates and children at risk for pressure injuries
- discuss challenges in neonatal and pediatric skin and wound care.

Introduction

In the past decade, there has been heightened awareness of the challenge of neonatal and pediatric pressure injury development in acute care patient population.[1,2] In addition, credentialing bodies such as Joint Commission and the American Nurses Credentialing Center (ANCC) Magnet Recognition Program have further highlighted the importance of identifying patients at risk for pressure injuries and instituting early interventions to prevent pressure injury development. Pressure injuries, even in the pediatric and neonatal settings, are considered a quality of care indicator and a largely preventable condition. Several years ago, a national network of children's hospitals called Children's Hospitals' Solutions for Patient Safety (SPS) and the Children's Hospital Association began collaborating to reduce harm by preventing specific hospital-acquired conditions, including pressure injuries.[3] The pressure injury work groups developed strategies consistent with high reliability concepts by creating prevention care bundles to reduce harm and improve patient outcomes. By 2018, there was a 26.3% reduction rate in hospital-acquired pressure injuries across the network hospitals.[3] Other benchmarking organizations include the NDNQI (National Database of Nursing Quality Indicators).[4]

Integumentary Development

Skin development is based upon gestational age.[2] The premature neonate, at 24 weeks' gestation, has minimal stratum corneum (2 to 3 layers compared to 10 to 20 layers in an adult), and rete ridges are very widely spaced thin and smaller in number.[2] Premature neonates are born with red, wrinkled, translucent skin that is almost gelatinous in appearance. Subcutaneous tissue has not yet developed, so the dermal tissue (dermis) is lying over the muscle. As subcutaneous fat deposit begins to form (weeks 26 to 29), skin wrinkling starts to decrease. Maturity of the integumentary system is complete at approximately 33 weeks' gestation. As the infant continues to develop (40 weeks' gestation), the stratum corneum begins to increase in thickness. The skin remains extremely fragile and easily insulted due to developmental immaturity. Given that the skin continues to develop through the first year of life, the use of appropriate, evidenced-based skin care practices is important.[5] Many factors predispose hospitalized infants to skin injuries, including decreased epidermal–dermal cohesion, deficient stratum corneum, relatively alkaline pH of skin surface, impaired nutrition, and presence of multiple medical devices secured on the skin. Epidermal stripping, extravasation injuries, incontinence-associated dermatitis, and pressure injuries are some of the most common injuries seen in the neonatal and pediatric population. Some authors include epithelial stripping from adhesive-based products as pressure injuries while others do not, thus further confounding pressure injury benchmarking data.[6] This chapter focuses mainly on pressure injuries that occur in a heterogeneous hospitalized neonatal and pediatric population.

Practice Point

Pressure injuries in pediatric patients are attributed to skin immaturity and body size.[2]

Pressure Injury Prevalence and Incidence

Published pressure injury incidence and prevalence figures in the acute care pediatric population vary widely. Prevalence data can be difficult to analyze and utilize as a benchmark due to methodological limitations and insufficient level of reporting detail (i.e., patient ages, critical care vs. noncritical care, surgical wards vs. medical wards, traditional immobility–related etiology vs. medical device–related, and/or variances in pressure injury reporting), although this is improving with the increased availability of benchmarking data nationally.[6,7] Also, there are often incomplete or unclear descriptions of inclusion and exclusion criteria making it difficult to generalize and benchmark findings. Overall, pediatric pressure injury prevalence rates during the past decade have been reported as low as 0.1% to 2.3%,[1,4] to as high as 27.7% to 35%.[8–10] In 2017, Razmus and Bergquist reported a descriptive secondary analysis of 2012 data submitted to the National Database for Nursing Quality Indicators® from 271 hospitals on 39,984 patients ages 1 day to 18 years. Overall pediatric pressure injury prevalence rate was 1.4%, varying by unit type.[11] Reports of neonatal pressure injuries vary from a low of 12%[10] and 16%[11] to a high of 31.2%,[12] although authors combined and included skin injuries as a result of pressure, friction, shear, and epithelial stripping injuries as one prevalence rate.[13] Schindler et al.[14] reported on data analyzed from over 5,300 pediatric intensive care unit (PICU) patients with reported 10.2% pressure injury prevalence. Noonan et al.[15] reported a 27% in three PICUs, and Schlüer et al.[10,16] reported 44% PICU prevalence. Prevalence rates of 20% have been reported in PICUs and neonatal intensive care units (NICUs), respectively, with the majority of these injuries occurring within 2 days of admission.[15–17] Schindler and colleagues[14] studied 372 pediatric ICU patients between the ages of 0 and 3 months and found an overall pressure injury incidence of 18.8%. It is important for facilities to track their own prevalence and incidence data to follow trends over time, influence educational initiatives, and maintain quality. There are no recent

studies that have been published on pressure injury prevalence in acute care pediatric settings specifically on special patient populations with neurosensory impairment, spine deformities, and postural challenges such as muscle spasticity or flaccid paralysis as seen with cerebral palsy or myelomeningocele (“spina bifida”). One could expect that the incidence and prevalence of pressure injuries in these specific patient populations would be even higher. It is important that clinicians pay particular attention to these patient populations due to frequent use of orthotics, wheelchairs, and other medical devices. Routine inspection for the proper fit of devices due to the normal trajectory of growth needs to be anticipated during childhood. This is particularly important during hospitalizations when they may have surgical procedures resulting in perioperative body temperature variability, hemodynamic changes, postop positioning challenges, increased incontinence, and edema from fluid volume resuscitation.

Anatomical Distribution

The occipital area is noted as being the most prevalent area for skin breakdown in children[1,2] (Figs. 25-1 and 25-2). Multiple authors have identified the occiput as the most common anatomical site for immobility-related pressure injury formation in patients from birth to age 3, as the head comprises a disproportionately higher percentage of the total body weight and surface area.[18–23] When lying supine, the occiput is the primary pressure point with the greatest interface pressure.[24] The EPUAP, NPIAP, PPPIA 2019 Clinical Practice Guideline has included the following implementation consideration based on expert opinion "Conduct a head-to-toe assessment with particular focus on skin overlying bony prominences, including the sacrum, heels, hip, pubis, thigh, and torso. Include the occiput in a head-to-toe skin assessment for neonates and young children."[1] Curley and colleagues[18,19] report the ear, the occiput,[24] and the nose[23] are the common sites for pressure injury development in pediatric patients. The nose is at greatest risk for medical device–related pressure injuries (MDRPIs) when noninvasive ventilation (NIV) masks, such as when used with continuous positive airway pressure (CPAP), are in use.[25,26] In this area, there is little tissue between the skin and the bone, and full-thickness injuries can develop quickly.[27] Additionally, mucous membranes such as the inside of the nare and the edge of the lip are susceptible to device-related injuries.[26,27] Willock and colleagues[28] conducted a multicenter survey in 11 hospitals and report the most frequent site of pressure injury development was the sacrum or buttocks, heel, thigh, ear, occipital scalp, malleolus, and spine. The ears do not have adipose tissue and therefore are also at increased risk when oxygen nasal cannula tubing or NIV masks are in place. Kottner and colleagues[8] completed a pediatric literature review and found the head including occiput, ears, and neck were most often affected followed by heels and sacral and ischial areas. August et al.[6] studied 247 neonates and reported that the head region had 35.5% of pressure injuries. However, it is important to take the age and size of the child into consideration. A large immobile child may very well have

a higher pressure interface at the sacrum than at the occiput, similar to adults, especially with prolonged head of bed elevation.[29]

Figure 25-1. Stage 2 pressure injury on an infant's occiput due to immobility.

Figure 25-2. Unstageable pressure injury on an infant's occiput due to immobility.

Practice Point

The occiput is the most common site for immobility-related pressure injuries in children from birth to age 3.

Risk Factors

Pressure injury development had traditionally been viewed as uncommon among neonatal and pediatric populations given the presumed relative ease of repositioning and frequency of movement.[8,29–31] However, as survival rates among critically and chronically ill premature neonates and children increase through technological advances, so too does the risk for pressure injury formation.[31] Premature neonates also have a higher ratio of skin surface area to weight with decreased or absent subcutaneous fat development based upon gestational age. Their anatomically immature skin is further challenged by prolonged intensive care to manage respiratory inefficiency, inadequate tissue perfusion, fluid and electrolyte issues, poor nutrition, and thermal regulatory issues that further increases risk for negative physiological responses to repositioning and pressure injury development. Infant skin has a higher absorption rate as compared with an adult's skin. This difference in the absorption rates also predisposes infants to a dry, flaky, and impaired skin barrier.[31,32] Analogous to adults, critically ill and injured neonates and children may experience massive edema secondary to fluid resuscitation and hemodynamic, respiratory, renal, and neurologic instabilities that increase the risk for an unavoidable PU.[33] Prolonged periods of immobilization may be needed, especially among those on extracorporeal membrane oxygenation (ECMO) and high-frequency oscillatory ventilation (HFOV).[34,35]

Furthermore, pediatrics cares not only for infants and children but for many adolescent and young adults as well. Among pediatric hospitals, there exist variable age-limit policies; therefore, a wide variety of patients are cared for, from neonates to obese adolescents and young adults to middle-aged patients. This poses additional challenges for clinicians to maintain clinical expertise in a variety of support surfaces and skin care products to manage a widely diverse patient populations seen in their facilities. There are two risk factor recommendations specific for neonates and children in the 2019 EPUAP, NPIAP, PPPIA 2019 Clinical Practice Guideline[1] (see Box 25-1).

Box 25-1

Recommendation	Strength of Evidence[a]	Strength of Recommendation[a]
"1.19: Consider the impact of skin maturity, perfusion and oxygenation, and presence of a medical device on pressure injury risk in neonates and children."	B1	↑↑
"1.20: Consider the impact of illness severity and the duration of critical care unit stay on pressure injury risk in neonates and children."	B2	↑

[a]See *Appendix EPUAP/NPIAP/PPPIA 2019 Clinical Guideline Strength of Evidence and Strength of Recommendation for definition of strength of evidence and strength of recommendation used in this guideline*.

Medical Equipment or Devices

For neonates and young children, devices including medical devices are a leading cause of pressure injuries.[1,2] Increasing numbers of critical access and monitoring lines/catheters and other forms of medical equipment often limit safe turning and repositioning options and increase the risk for unavoidable pressure injuries.[19,34,35] In critical care environments, positioning of the head is often limited by internal and external jugular vascular access catheters to avoid inadvertent catheter dislodgement, head and neck edema, and potential for air leak around endotracheal tubes with movement. Medical devices and monitoring equipment secured to the skin or mucosal membranes and compressing tissue are now widely recognized as a risk factor for pressure injury development. This is particularly a concern in pediatrics as young active patients or those with developmental impairment do not cognitively understand not to touch or play with equipment; therefore, clinicians tend to overly secure and stabilize devices to the skin to avoid inadvertent dislodgement. Data show that up to 65% to 71% of all pressure injuries in the acute-care pediatric setting were device related.[19,25,27,36] Previously, Willock and colleagues[28] found that 50% of neonatal and pediatric pressure injuries were directly associated with equipment pressing on the skin. Boyar (2018) reports the most common devices included respiratory devices, peripheral intravenous catheters and central lines, tracheostomies, and other devices[36] (Table 25-1). Critically ill patients typically require a multitude of invasive and noninvasive equipment anchored securely to their skin placing them at particularly high risk. Specific devices that may result in pressure injuries are respiratory devices (endotracheal/oral tracheal tubes, tracheostomy tube, NIV mask, and headgear), casts and orthotics, cervical spine immobilizers, pulse oximetry probes, and EEG leads to name a few (Figs. 25-3 to 25-8).

Table 25-1 Common Medical Devices That May Cause Pressure Injuries in Neonates and Children

- Oxygen saturation (O_2) probes
- Orthotics (casts, splints, braces, cervical collars)
- Respiratory devices (endotracheal/oral tracheal tubes, tracheostomy tube/flange, noninvasive ventilation masks/prongs, and headgear)
- EEG leads
- Arm boards
- Nasal cannula
- Gastrostomy/jejunostomy abdominal tube flanges or bolsters
- Cables, intravenous tubing
- Blood pressure cuffs
- Sequential compression devices (SCD)
- Nasogastric or orogastric tubes

Figure 25-3. Stage 1 pressure injury on an infant's nose caused by a medical device (BiPAP).

Figure 25-4. Unstageable pressure injury on an infant's occiput caused by a medical device (oxygen tubing).

Figure 25-5. Unstageable pressure injury on an infant's neck caused by a medical device (tracheostomy tube flange).

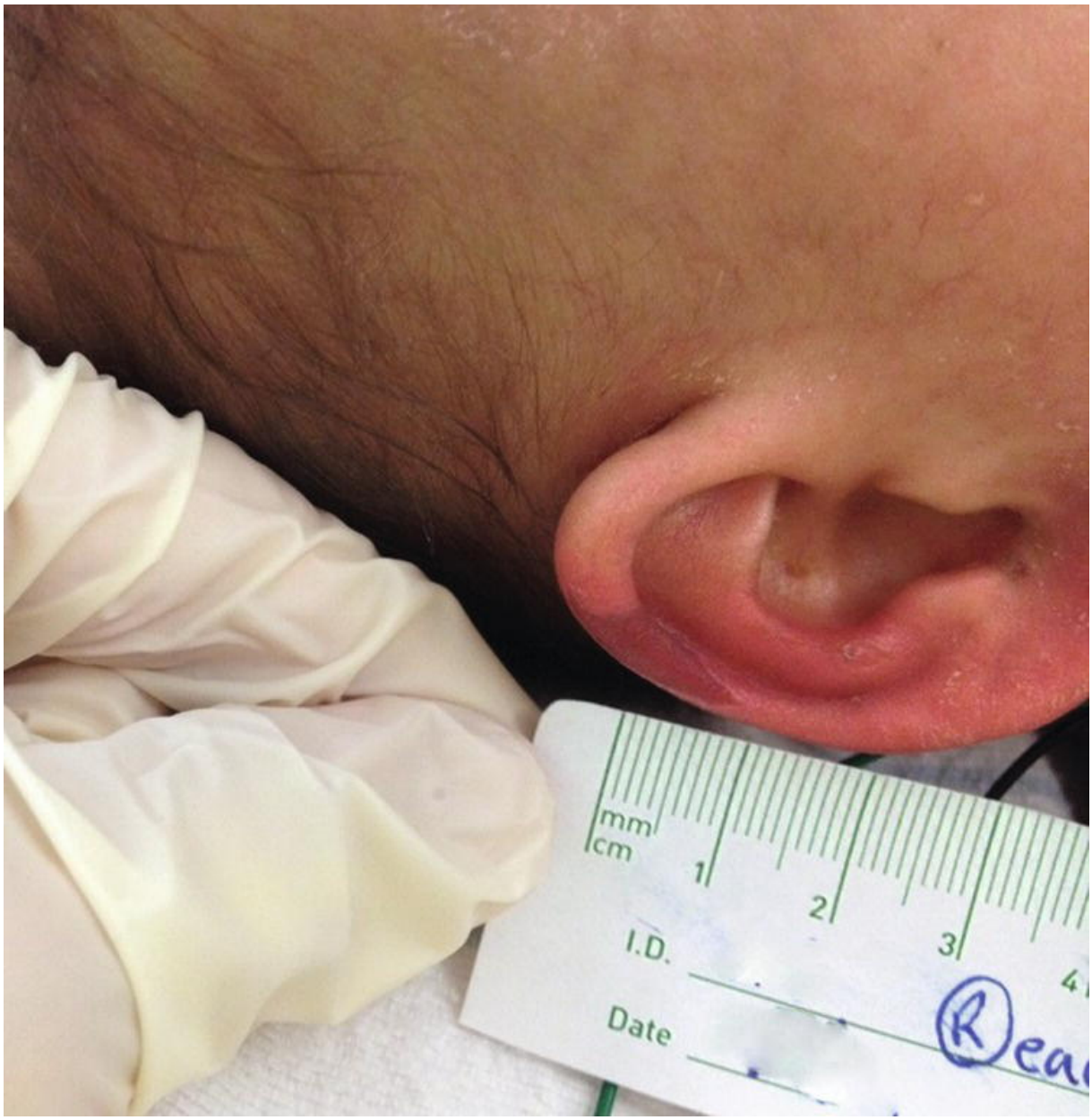

Figure 25-6. Suspected deep tissue injury right ear caused by a medical device (oxygen tubing).

Figure 25-7. Suspected deep tissue injury right great toe of an infant caused by a medical device (oxygen saturation probe).

Figure 25-8. Unstageable pressure ulcer injury on a child's left nasal nares caused by a medical device from a nasogastric device.

NIV therapy such as CPAP and bilevel positive airway pressure (BiPAP) devices place vulnerable neonates and children at risk for pressure injuries. These therapies and the associated devices have long been known to cause nasal injury and deformities.[37] The nasal-oral mask or full-face mask need to be properly fitted and secured snugly but not too tight to the skin to ensure adequate "seal." The high risk for ala (lateral surface of external nose that forms outer wall of each nostril), caudal septal, columnar, and nasal bridge pressure injury, which can occur secondary to nasal prongs and/or CPAP mask

and headgear, is of concern in all NICUs.[25,38,39] In addition to pressure, NIV masks can also alter the microclimate and encourage excessive moisture in the area, also contributing to pressure injury development.[38] Fischer et al. reported a 42.5% incidence of nasal trauma rate on 989 patients who were treated with nasal CPAP in NICU with 90% occurring in first 6 days of the therapy.[26]

Best practices for preventing MDRPIs include correct size of device, inspecting the skin in contact with the device at least daily, recognizing the impact of edema around devices, and cushion and protect skin in high-risk areas before skin issues occur.[27] Device-specific preventive measures, such as hourly prong or mask repositioning with skin assessments and the use of protective hydrocolloid or foam dressings, often prevent the occurrence of NIV injuries.[25,39,40] The EPUAP, NPIAP, PPPIA 2019 Pressure Injury Guideline[1] has a specific recommendation for neonates, which states "If appropriate and safe, alternate the oxygen delivery device between correctly fitting mask and nasal prongs to reduce the severity if nasal and facial pressure injuries for neonates receiving oxygen therapy. (Strength of Evidence = B1; Strength of Recommendation = ↑.) Murray and colleagues[41] recommend changing oxygen saturation probe locations every 2 to 3 hours (consider more frequently in critical care areas), removing orthotics two to three times daily and as needed, and changing dressings under tracheostomy and enteral tube sites one to two times daily to ensure proper fit and a moisture-free environment, as well as to relieve the pressure. If these actions are medically or clinically contraindicated, this should be documented.

In a case-controlled study of 118 PICU patients by McCord and colleagues, edema, PICU length of stay longer than 96 hours, increased positive end-expiratory pressure (PEEP), weight loss, and not turning the patient or using a specialty bed in turn mode were all identified as risk factors for pressure injury development.[42]

Special Populations

Paralysis and insensate skin related to spinal cord injury or myelomeningocele ("spina bifida") have been shown to be risk factors for pressure injury development in pediatric patient populations.[43,44] Ekmark[43] reported that shear injuries can occur during wheelchair transfers or transferring out of a car on warm days. Patient and family education essential regarding need to reposition at least every 1 to 2 hour when in wheelchair and to have specialized seat cushion reassess by a professional seating specialist annually and after any spine or lower extremity orthopedic surgery as pressure point areas may be different. Ekmark[43] suggests that new orthotic braces should be checked regularly, particularly 2 and 12 months, and also, whenever children gain or lose weight, wheelchair equipment must be modified. When new orthotic applied clinicians must ensure correct fit and educate patient and caregivers to do daily skin checks to identify early skin compromise, regularly cleanse the skin and allow skin to dry prior to application of orthotic. If possible, the first few days after a new orthotic applied alternate a few hours on then remove to check skin and allow skin to rest then reapply. Schottler and colleagues[44] completed a 27-year retrospective chart review of children who had spinal cord injuries at 5 years of age and younger and reported 41% had a history of a pressure injury with over 70% located in the foot and ankle. In a retrospective, exploratory study by Samaniego of 69 pediatric outpatients with a primary diagnosis of myelodysplasia, paralysis, insensate areas, high activity, and immobility were all identified as pressure injury risk factors.[45]

Risk Assessment Tools

To utilize objective criteria to determine patient risk, quantify the severity of risk, and guide clinicians to institute interventions that prevent skin compromise, the use of a pressure injury risk assessment scale is indicated.[18] Patients at risk for pressure injury can have a much higher likelihood of an HAPI than patients determined not at risk.[46] Researchers have emphasized the importance of clinicians recognizing that risk assessment, prevention, and management of immobility-related and MDRPIs are different.[47] It is critically important to differentiate the two phenomena.[41] There are multiple published neonatal/pediatric pressure injury risk assessment scales.[22,48–57] Schumacher and colleagues[58] determined that available tools were too lengthy for clinical use and developed a short trigger tool to identify infants at risk for pressure injury development and performed it on 15 NICU patients and determined that although it did not quantify risk, it was an efficient initial screening tool. It is essential that risk identification occurs early to help prevent pressure injuries in neonates and children.[57] However, the only pediatric pressure injury risk assessment scales for which there are published sensitivity and specificity data are the Braden Q Scale,[56] Neonatal Skin Risk Assessment Scale (NSRAS),[19] Glamorgan Scale,[54] and the Braden QD Scale.[57]

The NSRAS was created in 1997 for neonates and modeled after the Braden Scale for Predicting Pressure Injury Risk in adults, although the original Braden Scale has never been psychometrically tested with any age group in the pediatric population.[59] NSRAS measures six subscales pertinent to neonates.[19] The subscales are based on validity testing among a small sample size of 32 NICU patients and demonstrated a sensitivity of 83% and a specificity of 81%.[19] The field of neonatology has changed over the last decade, necessitating a revision of the instrument to reflect evidence-based practice[60]; therefore, the original NSRAS was updated to incorporate the specialized humidification incubator standardly used today to manage

insensible water losses of premature infants with a pilot study to be conducted of the revised tool.

The Braden Q Scale, developed in the early 1990s for predicting pediatric pressure injury risk, is a widely used, valid, and reliable pediatric-specific pressure injury risk assessment tool that is currently available in nine languages.[55] In developing the Braden Q Scale, Quigley and Curley adapted the six subscale descriptions for the pediatric population and added a tissue perfusion and oxygenation subscale.[19,48] In a multisite prospective study of 322 PICU patients, which excluded children with intracardiac shunting or unrepaired congenital heart disease, the Braden Q Scale was found to be 88% sensitive and 58% specific at a cutoff score of 16.[19] When the Braden Q Scale was first published in 1996, Quigley and Curley[48]used a cutoff of 8 years of age because at the time, the American Heart Association used that cutoff age for pediatric versus adult resuscitation standards. The Braden Q contains all the dimensions of the original Braden Scale, and it is widely used in neonates through adulthood. The Braden Q incorporates the developmental needs of pediatric patients, the prevalence of gastric/transpyloric tube feedings, use of available blood studies, and noninvasive technology. The Braden Q Scale[48] has a "tissue perfusion and oxygenation" subscale to quantify risk associated with compromised tissue perfusion. The original study excluded those with complex congenital heart disease so as not to confound the tissue perfusion and oxygenation scale with acutely ill patients who have compromised baseline oxygen saturation levels. Of note, the Braden Q was not designed to predict medical device–related injuries.

More recently, the authors have been coinvestigators in an IRB multicenter pediatric study to establish the reliability and validity of the Braden QD Scale (Fig. 25-9) and no longer recommend using the Braden Q Scale, rather the use of the Braden QD Scale is recommended. The prospective cohort study was performed in 8 centers, enrolling 635 patients, to provide data for Braden Q Scale revision and new tool development. The Braden QD Scale was published in 2017, and it is a single risk assessment scale to assess both immobility-related and device-related pressure injury in the patient populations typically cared for in a children's hospital, including those with congenital heart disease who were excluded from the original

Braden Q study. The scale was validated in preterm infants to age 21 years who were on bedrest for at least 24 hours and had a medical device in place. It is similar to the Braden Q Scale, although two of the subscales were removed, two device-related subscales added, and the scoring options adjusted. All patient types (surgical, medical, critical care and noncritical care, neonatal, and cardiovascular) were included in the validation study. With a cutoff of score of 13, the Braden QD Scale was found to have a sensitivity of 88% and specificity of 58%.[57] Since its publication, many hospitals are expressing interest in using the Braden QD Scale, and information on nurse education and integrating the scale into the facility is already available.[61,62]

Braden QD Scale				
Intensity and Duration of Pressure				**Score**
Mobility The ability to independently change & control body position	**0. No Limitation** Makes major and frequent changes in body or extremity position independently.	**1. Limited** Makes slight and infrequent changes in body or extremity position **OR** unable to reposition self independently (includes infants too young to roll over).	**2. Completely Immobile** Does not make even slight changes in body or extremity position independently.	
Sensory Perception The ability to respond meaningfully, in a **developmentally** appropriate way, to pressure-related discomfort	**0. No Impairment** Responsive **and** has no sensory deficits which limit ability to feel or communicate discomfort.	**1. Limited** Cannot always communicate pressure-related discomfort **OR** has some sensory deficits that limit ability to feel pressure-related discomfort.	**2. Completely Limited** Unresponsive due to diminished level of consciousness or sedation **OR** sensory deficits limit ability to feel pressure-related discomfort over most of body surface.	
Tolerance of the Skin and Supporting Structure				
Friction & Shear ***Friction:*** occurs when skin moves against support surfaces ***Shear:*** occurs when skin & adjacent bony surface slide across one another	**0. No Problem** Has sufficient strength to completely lift self up during a move. Maintains good body position in bed/chair at all times. Able to completely lift patient during a position change.	**1. Potential Problem** Requires **some** assistance in moving. Occasionally slides down in bed/chair, requiring repositioning. During repositioning, skin often slides against surface.	**2. Problem** Requires **full** assistance in moving. Frequently slides down and requires repositioning. Complete lifting without skin sliding against surface is impossible **OR** spasticity, contractures, itching or agitation leads to almost constant friction.	
Nutrition *Usual* diet for age – assess pattern over the most recent 3 consecutive days	**0. Adequate** Diet for age providing **adequate** calories **&** protein to support metabolism and growth.	**1. Limited** Diet for age providing **inadequate** calories **OR inadequate** protein to support metabolism and growth **OR** receiving supplemental nutrition any part of the day.	**2. Poor** Diet for age providing **inadequate** calories **and** protein to support metabolism and growth.	
Tissue Perfusion & Oxygenation	**0. Adequate** Normotensive for age, **&** oxygen saturation ≥ 95%, **&** normal hemoglobin, **&** capillary refill ≤ 2 seconds.	**1. Potential Problem** Normotensive for age **with** oxygen saturation <95%, **OR** hemoglobin <10 g/dl, **OR** capillary refill > 2 seconds.	**2. Compromised** Hypotensive for age **OR** hemodynamically unstable with position changes.	
Medical Devices				
Number of Medical Devices	**Score 1 point for each medical device* up to 8 (Score 8 points maximum)** ****Any diagnostic or therapeutic device that is currently attached to or traverses the patient's skin or mucous membrane.***			
Repositionability/ Skin Protection	**0. No Medical Devices**	**1. Potential Problem** All medical devices can be repositioned **OR** the skin under each device is protected.	**2. Problem** Any one or more medical device(s) can**not** be repositioned **OR** the skin under each device is not protected.	
			Total **(≥ 13 considered at risk)**	

© Curley MAQ; Adapted with permission from B. Braden and N. Bergstrom, Braden Scale for Predicting Pressure Sore Risk, (1987)

Figure 25-9. The Braden QD Scale.

(From Curley, M.A.Q., Hasbani, N.R., Quigley, S.M., et al. Predicting Pressure Ulcer Risk in Pediatric Patients: The Braden QD Scale. *The Journal of Pediatrics* 192:189-95, 2018.)

Another available risk assessment scale that includes medical device–related risk is the Glamorgan Scale. The Glamorgan Scale was developed by identifying risk factors through literature reviews and using the opinions of clinical experts and is based on factors thought to result in the development of pressure injuries in pediatric patients. Scoring is in two categories mobility and equipment with a weighted risk value score of 15 if there is any equipment pressing on skin. Any patient who scores a 15 or higher is determined to be at "high risk." When used in the assessment of pressure injury risk in 336 patients (1 day to 18 years of age), the Glamorgan Scale was found to be 98.4% sensitive and 67.4% specific at a cutoff score of 15.[56,57,63] The higher the Glamorgan total score, the greater the pressure injury risk: ≥10 = at risk, ≥15 = high risk, and ≥20 = very high risk.[53,57,63] In an observational validation study in a pediatric cardiac unit by Kottner and colleagues,[64] they found the Glamorgan scale was unable to make clear distinctions in a low-risk setting; therefore, it is unlikely the tools in this setting provide additional information for clinical decision-making.

A further challenge for clinicians is capturing the pressure injury risk to patients during often lengthy procedures in the operating room, radiology suite, or cardiac catheterization lab. It is imperative for nurses, surgeons, and anesthesiology colleagues to collaborate and assess risk for pressure injury development, specific to the positioning needs related to surgical procedures as well. Positioning will vary based upon specific surgical procedures, such as prone position for posterior spinal fusion, and require skin assessment on the frontal surface, which was gravity dependent during surgery. Surgical patients need to have their skin assessed during the perioperative period and any areas of skin compromise should be included in the patient "hand off" between care areas such as operating room to recovery room. The incidence and prevalence of pressure injuries for inpatients have been reported, but the effect of surgical procedures on pressure injury prevalence and development during admission has not been recognized. Current pressure injury risk tools do not address the patient who comes in for surgery, is admitted to the hospital after surgery, and then develops a pressure injury.[19,56] Galvin and Curley conducted a comprehensive review of pressure-related skin injuries

reported in 24 operative suites and then did a literature review to identify risk assessment scales and intraoperative pressure injury prevention strategies for pediatric patients. Consistent with the Braden Q Scale, Galvin and Curley used Braden and Bergstrom's conceptual framework on the etiology of pressure injuries to organize a pressure injury prevention bundle and developed the Braden Q + P Pressure Injury Risk Assessment Tool with a yes/no scoring system for each subscale.[56]

Practice Point

Use a pressure injury risk assessment scale designed for pediatric patients.

Pressure Redistribution Products

Historically, infants and children were placed on support surfaces (mattresses and overlays) originally designed for adults, the clinical efficacy of many products with patients of lower weights and smaller stature is unclear. Also, variability of number of layers of linen and continence pads placed by caregivers under patients poses an additional challenge. Many support surface manufactures will disclose upper body weight capacity but not minimal weight requirements. Milbrath and colleagues[65] reported that comparative data demonstrating product effectiveness with persons weighing less than 70 pounds, pressure distribution qualities of construction materials, support for positioning, impact on bed motility, and floatation under load at pressure points were missing. They further reported that patients and their families value comfort and quiet operation and expressed dissatisfaction with powered specialty mattresses due to the operating cyclic on-and-off noise.[65] Premature neonates do not have the muscle tone or strength that full-term infants have and, when placed on their backs, may lie in an extended position causing atypical neuromotor development. To promote flexion patterns of movement, caregivers need to routinely reposition patients using external support aides such as gel or fluidized positioners that facilitate the curled, flexed intrauterine position. Integral to pediatric education is the recognition of each child's uniqueness, the developmental characteristics of each age group, and the psychological and psychosocial factors they face.[19,28]

Today, clinicians have a wide variety of both powered and nonpowered options: isolette, crib, incubator, pediatric foam, and foam-/gel-topped mattresses and overlays available to choose from as compared to a decade ago. Because children are not "small adults," they should not be placed on adult support surfaces but instead on pressure redistribution products specifically designed for their specialized loading needs. Low–air loss beds designed for adults do not have the numerical option to accommodate for the height and weight of an infant or small child.[17] Children and infants often sink in and between cushions.[17] When adult specialty beds are placed in the

turn mode, the occiput of small children pivots on the same pressure point, potentially increasing shear and friction and not redistributing pressure.[42] If a low–air loss bed or alternating overlay is clinically indicated, only those that are age and weight appropriate, clinically efficacious, and safe should be used in accordance with manufacturer's recommendations. There are many, many companies who manufacture a large variety of pressure redistribution surfaces; the clinician must be vigilant to research any rental or purchased products that may be brought into their facility. Much more comparative research and evidence are needed to examine more closely the use of the various therapies in the nonadult populations.[66] The National Pressure Injury Advisory Panel created a Support Surface Standards Initiative called S3I in the hope to develop uniform terminology, test methods, and reporting standards for support surfaces. Check out the NPIAP Web site for more information on S3I.

A pilot study of 54 children at Texas Children's Hospital in 2002 by McLane et al. found that the highest interface readings were on the occiput from infancy to age 6 and on the occiput, coccyx, and heels from ages 6 to 18.[67] For children younger than age 2, use of the Delta foam overlay (Span America, Greenville, SC) resulted in the lowest interface occiput pressures.[67] For children over age 2, use of the Delta foam overlay with a Gel-E Donut head support and positioning aid (Philips Healthcare, Andover, MA) significantly lowered occipital pressure and provided generalized pressure redistribution.[67] Another nonpowered fluidized positioning aide (Mölnlycke® Z-Flo™ Fluidized Positioners, Mölnlycke®, Norcross, GA) is used to redistribute force over a greater surface area or to help offload bony prominences while maintaining neutral body alignment. Once molded to meet the anatomic needs of an individual patient, they will hold that shape until remolded. They conform to and support the patient's body in a comfortable, therapeutic position until next repositioning and are available in a range of sizes. The positioners are widely used in NICUs and in a variety of pediatric environments. More recently, a complete mattress replacement system designed for the pediatric population (Pediatric Pulse™, Sizewise, Lenexa, KS) is available for patients weighing between 0.7 and 45 kg. It offers a three-zoned support surface and has vented air cells that provide the option of either static

therapy or pulsating low air loss, as well as manage microclimate. Air-fluidized products Air-fluidized products include the Cliniton® or the Envella™ air fluidized beds (Hill-Rom, Batesville, IN). A product that simulates the effects of a body floating in a fluid and reduces vertical shear is the Dolphin System (Joerns, Arlington, TX), which applies fluid immersion simulation technology originally developed to transport US Navy dolphins over long distances in a dry environment. Early clinical use was in the perioperative environment as a surgical overlay system for the operating room table, where the Dolphin Pad can be used to prevent pressure injuries from lengthy surgical procedures.

Practice Point

Clinicians should know manufacturer disclosures to ensure that product materials, particularly foam cellular pads and mattresses, meet their state- or city-approved flame-retardant fire regulation compliance standards. Also, when using mattress overlays, consideration of mattress height versus siderail height must be taken into account to prevent patient falls.

Skin Care

Considering that pressure injuries occur under a plethora of medical devices, it is imperative that frequent, comprehensive skin inspection occur under all removable devices unless medically contraindicated. To complicate the situation further, materials that might be used to secure medical devices (e.g., tape, straps, dressings) may prevent assessment of adjacent skin and increase the risk for pressure injury development.[41] It is particularly important to assess skin under devices when a patient is developmentally unable to communicate discomfort and has neurosensory impairment such as those with chemical paralysis or spinal cord injury, as well as those with fluid resuscitation that results in localized or systemic edema, which may affect proper fit of medical device. Given that upward of 50% of pressure injuries are related to pressure from equipment and devices,[28] it's important to perform frequent skin assessments, rotate blood pressure cuffs and transcutaneous oxygen tension ($tcPO_2$) probes, and provide sufficient padding under tracheostomy plates, nasal prongs, CPAP and BiPAP masks, arm boards, orthotics/casts, and traction boots. All clinicians are responsible for monitoring skin and mucous membranes for alterations in skin integrity. Providers of care including nurses, clinical assistants, physicians, and therapists play an essential role in assessing, preventing, and managing pressure injuries.[68,69] Collaboration between nursing and respiratory therapy (RT) to move endotracheal tubes and NIV BiPAP or CPAP devices to assess for pressure injuries of the nose, lips, cheeks, or mucosal injuries of the tongue and buccal mucosa would help prevent or identify concerns early.[41] Manufacturers of NIV equipment today offer variety of masks, headgear, pillows, and chin straps giving clinicians alternative interfaces to avoid skin breakdown in infants and children. Implementing prevention strategies to ensure proper fitting of orthotics, transabdominal feeding tube flanges, wheelchairs, and wheelchair cushions as children grow must be integrated into all clinician practices. Applying interface layers such as foam and gel dressings/products between devices and skin to protect the skin and redistribute pressure should routinely be incorporated into bedside

practice before skin compromise occurs. Moisture-wicking fabrics can be used around tubes, skin folds, and surgical sites to help moisture wick away from the skin.[70]

Beds and cribs should be inspected to ensure that no tubing, cables, leads, hard toys, or syringe caps are inadvertently left under the patient's skin.[26,66] Tapes securing nasogastric and orogastric tubes, head dressings, and hats should be gently removed and the underlying skin assessed for pressure injuries. Routinely scheduled removal of EEG monitoring pads to assess skin on scalp and forehead should be coordinated with EEG technologist and bedside nursing.

Practice Point

Best Practices for Prevention of Medical Device–Related Pressure Injuries in Pediatric Populations Poster[71] is available for free download at https://www.npuap.org/wp-content/uploads/2013/04/Updated-BestPractices-Pediatric2017.pdf.

Practice Point

Because almost half of pressure injuries in children occur from tubes and devices, device repositioning, skin protection, and routinely assessing the skin beneath devices when clinically able are critical to identify skin compromise and prevent breakdown.

Nutritional Considerations

Nutrition and hydration are a concern to pediatric nurses who must balance their patient's medical condition nutrient needs with the ongoing dietary demands of growth. Potential alterations in nutrition related to metabolic deficiencies, congenital anomalies, swallowing disorders, inadequate intake, and/or increased metabolic need, as well as the additional challenges associated with toddler and school-age food preferences, are routinely encountered in pediatric care environments. The NPIAP published a white paper on the role of nutrition in pressure injury prevention and treatment for the adult patient population.[72] In 2019, they issued a white paper specific for the pediatric population.[2] The 2019 EPUAP, NPIAP, PPPIA 2019 Clinical guideline includes one good practice statement specific to the neonatal and pediatric population. It states "conduct age appropriate nutritional assessment for neonates and children at risk for pressure injuries."[1]

The systemic and immunological ramifications of malnutrition on compromised acute care patients further limit their tissue tolerance to pressure, friction, and shear, especially as third spacing occurs. Nutritional factors such as impaired intake, low birth weight, low body weight or unintentional weight loss, and dehydration may contribute to development of pressure injuries. It's essential that nutritional screening occurs during the admission assessment and that the protein, caloric, and hydration needs of neonates and children be addressed as part of a comprehensive pressure injury prevention and treatment plan. Patients need a weight, height, and BMI obtained and documented on admission so that weight trends can accurately be monitored. Care providers should collaborate with dietician to optimize nutrition plan and attempt to avoid confrontations around food choices to increase cooperation with selections. Offering 24/7 support for breast-feeding for those expressing an interest in breast-feeding is also paramount. Initiating the use of a breast pump as soon as it is determined that an infant cannot breast-feed or is made NPO should be standard of care. Furthermore, respect and awareness of vegetarian, gluten-free, peanut-free, lactose-free, and religious diets

must be incorporated into dietary choices offered to those who are able to tolerate oral intake in order to maximize nutritional intake.

Wound Care

When selecting a topical agent for use in the neonatal and pediatric populations, it is critical to consider the patient's age and degree of integumentary maturity, skin condition, product adherence, potential for skin sensitization, impact of product absorption, and need for avoidance of products containing dyes, fragrances, and preservatives.[29–31] Knowledge of product safety and manufacturer's recommendations in the neonatal and pediatric populations is essential.[29–31] Specific, neonatal skin and wound care recommendations are available in the updated Association of Women's Health, Obstetric and Neonatal Nurses (AWHONN), Neonatal Skin Care Evidence-based clinical Practice Guidelines, 4th edition[73] (available at www.awhonn.org).

Normal saline, sterile water, and gentle soap and water are all commonly used for wound cleaning in neonatal and pediatric populations. The selected wound care product or dressing should help to maintain an optimal wound healing environment by maintaining adequate moisture, humidity, pH, and temperature, by minimizing pain, and by preventing damage to the surrounding skin. Amorphous hydrogels and plain and silver hydrofibers can be utilized for pressure injury treatment. Thin hydrocolloids, foams, thin films, and silicone-based dressings may be used in both prevention and treatment.[74] If the wound bed has devitalized tissue or necrotic eschar and wound closure is the goal of treatment, then surgical or autolytic debridement should be considered unless patient is extremely medically fragile or on anticoagulant therapy. Although anecdotal case reports exist of topical enzymes having been used in the neonatal and pediatric populations, manufacturer recommendations for topical enzymes are only for those over age 18; safety and effectiveness in pediatric patients have not been established.[32] Medical-grade honey has been used effectively in both neonates and pediatrics. Silver-impregnated dressings are another consideration for children, but toxicity concerns do exist.[74]

Practice Point

Before using a topical agent, check with the manufacturer's guidelines to see if it is safe to use on the skin or wound of a neonate or pediatric patient.

In the treatment of extensive, full-thickness pressure injuries, negative pressure wound therapy (NPWT) may be utilized to achieve wound closure or as a bridge to surgical closure.[75,76] Stoffan and colleagues[75] reported on 18 infants who had abdominal wounds treated with NPWT and a median duration of treatment of 34.0 days and concluded that NPWT is an important therapeutic tool for the management of abdominal wounds in infants; however, there are some contraindications, including untreated osteomyelitis. NPWT technology remains without US FDA approval for patients under the age of 18.[75]

Pressure Injury Prevention Bundles

A care bundle incorporates those best practices that incorporate the critical components that if done in combination are likely to lead to better outcomes. The concept of bundling care practices is credited to the Institute for Healthcare Improvement.[77] Defined as a structured way of improving processes of care and patient outcomes, a bundle typically includes three to five evidence-based practices that when performed collectively and reliably, have been proven to improve patient outcomes.[77] Components of a pressure injury prevention program often include a comprehensive head-to-toe skin assessment within 8 hours of admission and then daily with a focus on bony prominences and skin under medical devices; use of a validated pressure injury risk assessment scale and then care planning and protocol implementation to address any areas of risk noted; and an audit tool done daily, weekly, or monthly to check compliance on elements of care. Auditing compliance with a prevention bundle can be done by combination of any of the following to monitor evidence of implementation: paper or electronic medical record documentation, visual observation, and/or skin inspection.

High compliance with a pressure injury prevention bundle has been shown to decrease pressure injury rates as much as 30% to 40%.[36] Elements of a pressure injury prevention bundle may include any or all of the following components:

- Skin assessment
- Pressure injury risk assessment
- Pressure redistribution/specialty bed surfaces and positioners
- Repositioning (per facility protocol)
- HOB elevated ≤30 degrees if medically not contraindicated
- Keep heels elevated off bed surface
- Moisture and incontinence management
- Skin assessment and care under/around medical devices (per facility protocol)
- Routine rotation of medical devices (if possible; per facility protocol)

- Prophylactic application of a sacral foam dressing to protect bony prominence

Patient and Family Education

To enhance quality of care, education regarding pressure injury risk factors, and prevention and management interventions is essential for all healthcare providers, as well as children and their families particularly those at high risk. Empowering families with knowledge will instill confidence and likely decrease risk of pressure injury occurrence not only during their hospitalization but at home once they are discharged. Teaching should review the following: avoid dragging or sliding child in and out of the chair or bed; avoid lying in same position and to reposition at least once an hour when sitting and every 2 hours when lying in bed; do not massage any skin that is red, warm or tender; keep skin clean and dry; and apply barrier ointments to protect skin from urine and stool. Parents should be taught to notify their child's nurse if they notice any reddened areas on the skin. When children have darker skin tones, teaching should reinforce to look for any areas of skin discoloration that may have purplish hue or feel warm to touch as compared to surrounding skin, particularly over bony prominences and under medical equipment. At the time of discharge, parents of high-risk infants and children should be taught to perform skin assessments during bathing, diaper changes, and application of orthotics or NIV equipment. Toddlers and preschool-age children can be taught how to perform skin checks on their doll or teddy bear and then on themselves.[36] School-age children with upper extremity abilities should be taught "liftoffs" with the use of mirrors to check the buttocks as well as how to ensure that their wheelchair cushion is functioning properly.[36] Written educational materials, alarm clocks or watches as reminders for liftoffs, and rewards for assuming self-care are beneficial. As parents begin to relinquish control, they should maintain a safety net role in their child's care.[36] Education of teenagers is best provided on a one-on-one basis with respect for their privacy. Educational materials that are concise and focused on their tasks are best received. Watches with automatic alarms to serve as a reminder for liftoffs are of benefit during this developmental stage as well.[78]

Practice Point

A pressure injury prevention teaching plan should be specific to the child's age, developmental level, and individual characteristics.

Summary

Based on available pressure injury prevalence and incidence data, hospitalized neonates and children are at risk for developing pressure injuries.[1,2,29–31] Neonates and children face unique problems from the development of pressure injuries. Pediatric care environments have an added challenge to maintain competence due to the diverse age population cared for from premature neonates through adults. Support surfaces and topical products manufactured for adults may not be suitable for use in neonates and children because their clinical efficacy and safety in these age groups are unknown. Neonates and children require skin care specific to their needs.[78] The development of much needed clinical practice guidelines and educational programs focused on pressure injuries in neonates and children must address the immature integument of the premature neonate, including the potential for absorptive toxicity and disparities in weight distribution, and the physiological and psychological uniqueness of these populations while acknowledging the basic tenets found in adult models.[29–31] The use of a risk assessment scale to identify patients at risk, in combination with a comprehensive skin assessment and clinical judgment, is key to a pressure injury prevention program. Furthermore, recognizing that any medical device increases a patient's risk for device-related pressure injuries is the first step in prevention.[71] Further research is needed into the risks of current technology and medical devices, with consideration for design changes to decrease the risk of pressure-related injuries from the devices themselves.[71] Further research is also needed to determine which pressure reduction interventions are the most efficacious in preventing HAPI.[79]

PATIENT SCENARIO

Patient is a 16-year-old male, being assessed 4 days after hospital admission for traumatic cervical spinal cord injury. He has no movement of his extremities. He has no sensation to light or firm touch to his extremities. He has a tracheostomy, with the tube sutured

in place and the original split gauze dressing in place with a planned first dressing change on day 5. His neck is moist from oral and stomal secretions, with reddened skin folds in the area of the tracheostomy securement device. His oxygen saturation via continuous monitor is 95% to 100% on room air. His vital signs are normal, and his hands and feet are cool to the touch (capillary refill time 3 seconds). He has an indwelling Foley catheter. He is on total parenteral nutrition providing him with adequate calories. A gastrostomy tube will be placed in several days.

Lab data in last 24 hours:

- Albumin 1.9 g/dL
- Hemoglobin 9 g/dL
- Serum pH from ABG = 7.35

Medical devices in place:

- Continuous cardiorespiratory monitor
- Continuous oximeter
- Tracheostomy tube/tracheostomy ties
- Peripherally inserted central catheter (insertion site secured with occlusive dressing, tubing stabilized to skin with tape)
- Bilateral multipodus boots

Using the Braden QD Scale (Fig. 25-9), *score the patient's pressure injury risk* for each subscale and for total score. What does the risk score mean? What interventions should be instituted?

- **Mobility**
- **Sensory perception**
- **Friction/shear**
- **Nutrition**
- **Tissue perfusion**
- **Number of medical devices**
- **Repositionability**

Case Discussion

Explanation of Scoring:

- Mobility: Patient is immobile. Scores 2.

- Sensory perception: Patient cannot feel sensation in extremities. Scores 2.
- Friction/shear: Patient requires full assistance in moving. Scores 2.
- Nutrition: Patient on total parenteral nutrition with adequate calories. Scores 1.
- Tissue perfusion: Patient has less than normal hemoglobin (9 g/dL) and delayed capillary refill time. Scores 1. Total score: 8.
- Number of medical devices: Patient has 7 devices: (a) cardiorespiratory monitor leads (group of leads counts as 1 device); (b) oximeter; (c) tracheostomy tube; (d) tracheostomy ties; (e) PICC line; and (f, g) Multipodus boots (each splint on an extremity is counted separately).
- Repositionability: The PICC line cannot be repositioned and the tubing is secured to the skin directly. The tracheostomy tube/ties cannot be adjusted per surgical team orders. Scores 2.

The patient's Braden QD Score is 17. This places him in the at-risk category for pressure injury development. Interventions should be focused on the at-risk subscales. Pressure redistribution support surface should be used and frequent turns and skin assessments since the patient has no sensation. Turning and lift devices should be used with lifting and position changes to prevent shear/friction to the skin. Nutrition should be followed closely, with adequate calories and proteins provided to meet caloric demands. Skin should be protected from all devices as able. In this scenario, there are several devices that cannot be repositioned or the skin padded underneath: the tracheostomy tube and ties, and the PICC line. Attention to padding and repositioning medical devices as able will be important.

Show What You Know

1. **Maturity of the integumentary system is complete at approximately weeks' gestation.**
    A. 40 weeks
    B. 28 weeks
    C. 33 weeks
    D. 36 weeks

2. **What is the most prevalent area for skin breakdown in neonates and children to occur?**
    A. Sacrum
    B. Ear
    C. Occiput
    D. Heels

3. **Which one of the following is a pressure injury risk assessment scale that can be used for a 5-year-old child.**
    A. Braden scale
    B. Waterlow scale
    C. Norton scale
    D. Braden Q scale and Glamorgan scale

Answers to these questions are found on page 688.

References

1. European Pressure Ulcer Advisory Panel, National Pressure Injury Advisory Panel and Pan Pacific Pressure Injury Alliance. Prevention and Treatment of Pressure Injuries/Injuries: Clinical Practice Guideline. The International Guideline 2019. In E. Haesler, ed. EPUAP/NPIAP/PPPIA, 2019.
2. Delmore, B., Deppisch, M., Sylvia, C., et al. Pressure Injuries in the Pediatric Population: A National Pressure Ulcer Advisory Panel White Paper. Advances in Skin & Wound Care 32(9):394-408, 2019.
3. Children's Hospitals' Solutions for Patient Safety. Available at: https://www.solutionsforpatientsafety.org. Accessed January 31, 2020.
4. National Data Base of Nursing Quality Indicators (NDNQI). Available at: http://www.pressganey.com/solutions/clinical-excellence/nursing-quality. Accessed January 31, 2020.
5. Telofski, L.S., Morello, A.P. III, Mack Correa, M.C., et al. "The Infant Skin Barrier: Can We Preserve, Protect, and Enhance the Barrier?" Dermatology Research and Practice 2012:1-18, 2012.
6. August, D.L., Edmonds, L., Brown, D.K., et al. "Pressure Injuries to the Skin in a Neonatal Unit: Fact or Fiction." Journal of Neonatal Nursing 20(3):129-37, 2014.
7. Wang, D., Xu, H., Chen, S., et al. Medical Adhesive-Related Skin Injuries and Associated Risk Factors in a Pediatric Intensive Care Unit. Advances in Skin & Wound Care 32(4):176-82, 2019.
8. Kottner, J., Wilborn, D., Dassen, T. "Frequency of Pressure Ulcers in the Paediatric Population: A Literature Review and New Empirical Data." International Journal of Nursing Studies 47(10):1330-340, 2010.
9. Schlüer, A.B., Cignacco, E., Müller, M., et al. "The Prevalence of Pressure Ulcers in Four Paediatric Institutions." Journal of Clinical Nursing 18(23):3244-252, 2009.
10. Schlüer, A.B., Schols, J.M.G.A., Halfens, R.J.G. "Pressure Ulcer Treatment in Pediatric Patients." Advances in Skin & Wound Care 26(11):504-10, 2013.
11. Razmus, I., Berquist-Beringer, S. Pressure Ulcer Prevalence and the Rate of Hospital-Acquired Pressure Ulcer Among Pediatric Patients in Acute Care. Journal of Wound, Ostomy, and Continence Nursing 44(2):110-7, 2017.
12. Burton, W., Broom, M., Kecskes, Z. Pilot Study on the Incidence and Cause of Skin Injuries in Neonates. Perinatal Society of Australia & New Zealand Hobart, Wiley-Blackwell, 2011:8-59.
13. Fujii, K., Sugama, J., Okuwa, M., et al. "Incidence and Risk Factors of Pressure Ulcers in Seven Neonatal Intensive Care Units in Japan: A Multisite Prospective Cohort Study." International Wound Journal 7(5):323-28, 2010.
14. Schindler, C.A., Mikhailov, T.A., Kuhn, E.M., et al. "Protecting Fragile Skin: Nursing Interventions to Decrease Development of Pressure Ulcers in Pediatric Intensive Care." American Journal of Critical Care 20(1):26-34, 2011.

15. Noonan, C., Quigley, S., Curley, M.A.Q. “Skin Integrity in Hospitalized Infants and Children: A Prevalence Survey.” Journal of Pediatric Nursing 21(6):445-53, 2006.
16. Schlüer, A.B., Halfens, R.J., Schols, J.M.G.A. “Pediatric Pressure Ulcer Prevalence: A Multicenter, Cross-Sectional, Point Prevalence Study in Switzerland.” Ostomy/Wound Management 58(7):18-31, 2012.
17. McLane, K.M., et al. “The 2003 National Pediatric Pressure Ulcer and Skin Breakdown Prevalence Survey.” Journal of Wound, Ostomy, and Continence Nursing 31(4):168-78, 2004.
18. Curley, M.A.Q., et al. “Predicting Pressure Ulcer Risk in Pediatric Patients: The Braden Q Scale.” Nursing Research 52(1):22-31, 2003.
19. Curley, M.A., et al. “Pressure Ulcers in Pediatric Intensive Care: Incidence & Associated Factors.” Pediatric Critical Care Medicine 4(3):284-90, 2003.
20. Schindler, C., Mikhailov, T., Cashin, S., et al. “Under Pressure: Preventing Pressure Ulcers in Critically Ill Infants.” Journal for Specialists in Pediatric Nursing 18:329-41, 2013.
21. Razmus, I.S., Roberts, K.E., Curley, M.A.Q. “Pressure Ulcers in Critically Ill Children: Incidence and Associated Factors.” Critical Care Medicine 29(Suppl A148), 2001.
22. Huffiness, B., Lodgson, M.C. “The Neonatal Skin Risk Assessment Scale for Predicting Skin Breakdown in Neonates.” Issues in Comprehensive Pediatric Nursing 20:103-14, 1997.
23. Manning, M.J., Gauvreau, K., Curley, M.A.Q. Factors Associated with Occipital Pressure Ulcers in Hospitalized Infants and Children. American Journal of Critical Care 24(4):342-8, 2015.
24. Solis, I., et al. “Supine Interface Pressure in Children.” Archives of Physical Medicine and Rehabilitation 69:524-26, 1988.
25. Jatana, K.R., Oplatek, A., Stein, M., et al. “Effects of Nasal Continuous Positive Airway Pressure and Cannula Use in the Neonatal Intensive Care Unit Setting.” Archives of Otolaryngology—Head & Neck Surgery 136(3):287-91, 2010.
26. Fischer, C., Bertelle, V., Hohlfeld, J., et al. “Nasal Trauma Due to Continuous Positive Airway Pressure in Neonates.” Archives of Disease in Childhood. Fetal and Neonatal Edition 95:447-51, 2010.
27. Miske, L., Stetzer, M., Garcia, M., et al. Airways and Injuries: Protecting Our Pediatric Patients From Respiratory Device-Related Pressure Ulcers. Critical Care Nursing Clinics of North America 29(2):187-204, 2017.
28. Willock, J., Harris, C., Harrison, J., et al. “Identifying the Characteristics of Children with Pressure Ulcers.” Nursing Times 101(11):40-3, 2005.
29. Baharestani, M.M., Ratliff, C.R. “Pressure Ulcers in Neonates and Children: An NPUAP White Paper.” Advances in Skin & Wound Care 20:208, 210, 214, 216, 218-20, 2007.
30. Baharestani, M.M., Pope, E. “Chronic Wound Care in Neonates and Children.” In D. Krasner, et al., eds. Chronic Wound Care: A Clinical Source Book for

Healthcare Professionals, 4th ed. Malvern, PA: HMP Communications, 2007.
31. Baharestani, M.M. “Neonatal and Pediatric Issues in Wound Care.” In J. McCulloch, L. Kloth, eds. Wound Healing: Evidence Based Management, 4th ed. Philadelphia, PA: FA Davis Company, 2010.
32. Baharestani, M.M. “Neonatal and Pediatric Wound Care: Filling Voids in Knowledge and Practice.” Ostomy/Wound Management 53(6):8-9, 2007.
33. Blume-Peytavi, U., Hauser, M., Stamatas, G.N., et al. “Skin Care Practices for Newborns and Infants: Review of the Clinical Evidence for Best Practices.” Pediatric Dermatology 29(1):1-14, 2012.
34. Edsberg, L.E., Langemo, D., Baharestani, M.M., et al. “Unavoidable Pressure Injury: State of the Science and Consensus Outcomes.” Journal of Wound, Ostomy, and Continence Nursing 41(4):313-34, July-August 2014.
35. Neidig, J.R., Kleiber, C., Oppliger, R.A. “Risk Factors Associated with Pressure Ulcers in the Pediatric Patient Following Open-Heart Surgery.” Progress in Cardiovascular Nursing 4(3):99-106, 1989.
36. Boyar, V. Outcomes of a Quality Improvement Program to Reduce Hospital-Acquired Pressure Ulcers in Pediatric Patients. Ostomy/Wound Management 64(11):22-8, 2018.
37. Robertson, N.J., McCarthy, L.S., Hamilton, P.A., et al. “Nasal Deformities Resulting from Flow Driver Continuous Positive Airway Pressure.” Archives of Disease in Childhood. Fetal and Neonatal Edition 75:F209-12, 1996.
38. Friedman, J. “Plastic Surgical Problems in the Neonatal Intensive Care Unit.” Clinics in Plastic Surgery 25(4): 599-617, 1998.
39. Smith, Z.K. “Adapting a Soft Silicone Dressing to Enhance Infant Outcomes.” Ostomy/Wound Management 52(4):30-2, 2006.
40. Chandler, A.J., Lewis, J., Hare, R. Medical Device-Related Pressure Ulcers in Premature Babies. Wounds UK 12(4):24-9, 2016.
41. Murray, J.S., Noonan, C., Quigley, S., et al. “Medical Device-Related Hospital-Acquired Pressure Ulcers in Children: An Integrative Review.” Journal of Pediatric Nursing 28(6):585-95, 2013.
42. McCord, S., McElvain, V., Sachdeva, R., et al. “Risk Factors Associated with Pressure Ulcers in the Pediatric Intensive Care Unit.” Journal of Wound, Ostomy, and Continence Nursing 31(4):179-83, 2004.
43. Ekmark, E.M. “Risky Business: Preventing Skin Breakdown in Children with Spina Bifida.” Journal of Pediatric Rehabilitation Medicine 2(1):37-50, 2009.
44. Schottler, J., Vogel, L., Sturm, P. “Spinal Cord Ulcers in Young Children: A Review of Children Injured at 5 Years of Age and Younger.” Developmental Medicine and Child Neurology 54:1138-43, 2012.
45. Samaniego, I.A. “A Sore Spot in Pediatrics: Risk Factors for Pressure Ulcers.” Pediatric Nursing 29(4):278-82, 2003.
46. Razmus, I. Factors Associated with Pediatric Hospital-Acquired Pressure Injuries. Journal of Wound, Ostomy, and Continence Nursing 45(2):107-16, 2018.

47. Kohr, L.M., Curley, M.A. “Small Study Finds 27% Prevalence of Pressure Ulcers in Paediatric Hospitals in Switzerland, with Many Cases Caused by External Medical Devices.” Evidence-Based Nursing 13:58, 2010.
48. Quigley, S.M., Curley, M.A.Q. “Skin Integrity in the Pediatric Population: Preventing and Managing Pressure Ulcers.” Journal for Specialists in Pediatric Nursing 1(1):7-18, 1996.
49. Bedi, A. “A Tool to Fill the Gap-developing a Wound Risk Assessment Chart for Children.” Professional Nurse 9(2):112-20, 1993.
50. Cockett, A. “Paediatric Pressure Sore Risk Assessment.” Tissue Viability Society 8(1):30, 1998.
51. Waterlow, J. “Pressure Sores in Children: Risk Assessment.” Paediatric Nursing 10(4):22-3, 1998.
52. Barnes, S. “The Use of a Pressure Ulcer Risk Assessment Tool for Children.” Nursing Times 100(14): 56-8, 2004.
53. Willock, J., Baharestani, M.M., Anthony, D. “A Risk Assessment Scale for Pressure Ulcers in Children.” Nursing Times 103(14):32-3, 2007.
54. Willock, J., Baharestani, M.M., Anthony, D. “The Development of the Glamorgan Paediatric Pressure Ulcer Risk Assessment Scale.” Journal of Wound Care 18(1):17-21, 2009.
55. Noonan, C., Quigley, S., Curley, M.A.Q. “Using the Braden Q Scale to Predict Pressure Ulcer Risk in Pediatric Patients.” Journal of Pediatric Nursing 26:566-75, 2011.
56. Galvin, P., Curley, M.A.Q. “The Braden Q + P: A Pediatric Perioperative Pressure Ulcer Risk Assessment and Intervention Tool.” AORN Journal 96(3):261-70, 2012.
57. Curley, M.A.Q., Hasbani, N.R., Quigley, S.M., et al. Predicting Pressure Injury Risk in Pediatric Patients: The Braden QD Scale. The Journal of Pediatrics 192:189-195.e2, 2018.
58. Schumacher, B., Askew, M., Otten, K. “Development of a Pressure Ulcer Trigger Tool for the Neonatal Population.” Journal of Wound, Ostomy, and Continence Nursing 40(1):46-50, 2013.
59. Murray, J.S., Quigley, S., Curley, M.A.Q. “Letter to Editor Re: Risk and Associated Factors of Pressure Ulcers in Hospitalized Children Over 1 Year of Age.” Journal for Specialists in Pediatric Nursing 19:105-6, 2014.
60. Dolack, M., Huffines, B., Stikes, R., et al. “Updated Neonatal Skin Risk Assessment Scale (NSRAS).” Kentucky Nurse 61(4):6, October-December 2013.
61. Braden QD Scale for Assessment of Immobility and Device Related Pressure Ulcer Risk in your Pediatric Population. Available at: http://www.marthaaqcurley.com/braden-qd.html. Accessed January 31, 2020.
62. Chamblee, T.B., Pasek, T.A., Caillouette, C.N., et al. How to Predict Pediatric Pressure Injury Risk with the Braden QD Scale. American Journal of Nursing 118(11):34-43, 2018.

63. Willock, J., Baharestani, M.M., Anthony, D. “The Development of the Glamorgan Paediatric Pressure Ulcer Risk Assessment Scale.” Journal of Children’s & Young People’s Nursing 1(5):211-18, 2007.
64. Kottner, J., Kenzler, M., Wilborn, D. “Interrater Agreement, Reliability and Validity of the Glamorgan Paediatric Pressure Ulcer Risk Assessment Scale.” Journal of Clinical Nursing 23(7–8):1165-9, 2014.
65. Milbrath, C., Linroth, R., Wilhelmy, J., et al. “A Method of Comparing Effectiveness of Mattresses for Pressure Management for Pediatric Patients.” Journal of Nursing Care Quality 29(1):66-73, 2014.
66. McNichol, L., Watts, C., et al. Identifying the Right Surface for the Right Patient at the Right Time: Generation and Content Validation of an Algorithm for Support Surface Selection. Journal of Wound, Ostomy, and Continence Nursing 42(1):19-37, 2015.
67. McLane, K., Krouskop, T., McCord, S., et al. “Comparison of Interface Pressures in the Pediatric Population Among Various Support Surfaces.” Journal of Wound, Ostomy, and Continence Nursing 29(5):242-51, 2002.
68. Apold, J., Rydrych, D. “Preventing Device-Related Pressure Ulcers: Using Data to Guide Statewide Change.” Journal of Nursing Care Quality 2:728-34, 2012.
69. Black, J., Cuddigan, J., Walko, M., et al. “Medical Device Related Pressure Ulcers in Hospitalized Patients.” International Wound Journal 7:358-65, 2010.
70. Singh, C.D. Use of a Moisture Wicking Fabric for Prevention of Skin Damage Around Drains and Parenteral Access Lines. Journal of Wound, Ostomy, and Continence Nursing 43(5):551-3, 2016.
71. Best Practices for Prevention of Medical Device–Related Pressure Injuries in Pediatric Populations. Poster is available for free download at https://www.npuap.org/wp-content/uploads/2013/04/Updated-BestPractices-Pediatric2017.pdf. Accessed January 31, 2020.
72. Posthauer, M.E., Banks, M., Dorner, B., et al. The Role of Nutrition for Pressure Ulcer Management. National Pressure Ulcer Advisory Panel, European Pressure Ulcer Advisory Panel, Pan Pacific Pressure Injury Alliance White Paper. Advances in Skin & Wound Care 28(4):175-88, 2015.
73. Association of Women’s Health, Obstetric and Neonatal Nurses (AWHONN). Neonatal Skin Care Evidence-based Clinical Practice Guidelines, 4th ed. Available at: www.awhonn.org. Accessed April 10, 2019.
74. King, A., Stellar, J.J., Blevins, A., et al. Dressings and Products in Pediatric Wound Care. Advances in Wound Care 3(4):324-34, 2014.
75. Stoffan, A., Ricca, R., Lien, C., et al. “Use of Negative Pressure Wound Therapy for Abdominal Wounds in Neonates and Infants.” Journal of Pediatric Surgery 47:1555-9, 2012.
76. Baharestani, M.M. “Use of Negative Pressure Wound Therapy in the Treatment of Neonatal and Pediatric Wounds: A Retrospective Examination of Clinical Outcomes.” Ostomy/Wound Management 53(6):16-25, 2007.

77. Resar, R., Griffin, F.A., Haraden, C., et al. *Using Care Bundles to Improve Health Care Quality*. IHI Innovation Series White Paper. Cambridge, MA: Institute for Healthcare Improvement, 2012. Available at: www.IHI.org
78. Hickey, K., Vogel, L.C., Anderson, C.J. “Pressure Ulcers in Pediatric Spinal Cord Ulcer.” Topics in Spinal Cord Ulcer Rehabilitation 6(Suppl):85-90, 2000.
79. Levy, A., Kopplin, K., Gefen, A. Adjustability and Adaptability Are Critical Characteristics of Pediatric Support Surfaces. Advances in Wound Care 4(10):615-22, 2015.

26

Palliative Skin and Wound Care

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Objectives

After completing this chapter, you'll be able to:

- define palliative wound care
- discuss palliative wound treatment
- delineate treatment for a fungating wound
- explain treatment for a radiation wound.

Defining Palliative Wound Care

Palliative care is an approach to enhancing quality of life for patients and their families facing a life-threatening condition and its associated problems. It is focused on preventing and minimizing suffering via early identification, assessment, and treatment of pain and suffering (http://www.WHO.int/cancer/palliative.definition/en/).[1] The 2002 National Consensus Project noted that palliative care is an organized and highly structured system that focuses care on promoting the greatest comfort for and dignity of the patient[2] and is best delivered by a multidisciplinary team.[3,4] (See Chapter 1, Quality of Life and Ethical Issues.) Palliative care programs are more difficult to have and sustain in rural areas. Telehealth, telemedicine, remote monitoring, and video conferencing are being used.[5] Centers for Medicare and Medicaid Services has plans to expand payment for these services.[5] In response to these national efforts, many hospitals in the United States and throughout the world have palliative care units or teams. However, in spite of these national-level studies and the growth in palliative care programs, public policies that focus on palliation need to be expanded.

The setting of palliative care, with patients who most often have multiple comorbidities and risk factors and who are at the end of life, is a high risk for pressure injury development.[4,6,7] Although the usual goal and plan of care is directed toward healing a wound, for individuals at the end of life (with a nonhealing wound), palliative wound care may be desired and the most appropriate goal. While often overlooked as the largest organ of the body, the skin can fail along with the other organs.[8] It is illogical to expect the skin to heal concomitant with the failure of other vital organ systems.[8,9] Most individuals, if not all, at the end of their lives are at risk for developing soft tissue ulceration.[4,10–18] Most professionals agree that pressure injuries occurring at the end of life are often unavoidable, largely attributable to the individual's frail, compromised condition, and often where an organ or organs are failing.[4,6,8,10,14,19–21] In fact, the literature is replete with reports that it is likely impossible to eradicate pressure injuries in end-of-life patients with their many comorbid conditions and risk factors.[4–

[7,11,14,18,22–29] However, with education and teaching, which should include a thorough question and answer session between the patient and family and the physician and other healthcare professionals, the goal of care should then be established. The decision to move a patient from a curative to a palliative treatment plan requires that the clinician has determined that the wound is ultimately nonhealing (rather than undertreated) and that palliation is consistent with the patient's goals.[4,29,30]

Extent of the Problem

Approximately 300 million individuals, or 3% of the world's population, are in need of end-of-life care every year, and in the United States it was approximately 2.5 million annually.[1,31] In 2020, that number would likely approach 3 million. Estimates are that by the year 2030, 20% of the U.S. population will be age 65 or older[14] and over 157 million Americans will suffer from chronic illnesses.[32,33] Given this demographic shift, a significant increase must be expected in the number of frail, elderly patients for whom cure may not be the goal. Overall, little information is available on wounds at the end of life.[4,18] Currently, however, wounds affect more than one-third of the nearly 1 million hospice patients in the United States, as well as many more patients at the end of life who are not under hospice care. Over 40 types of wounds have been identified in advanced illness.[18]

Relatively few studies on pressure injury prevalence and incidence in end-of-life patients appear in the literature, and, of those that do, most report on subjects with a cancer diagnosis. Little evidence on the prevalence and incidence of end-of-life wounds is available.[34,35] Pressure injury prevalence and incidence have been reported in nine studies worldwide, six in the United States, two in Europe, and one in Canada. Pressure injury incidence has varied from 6.5%[15] to 17%[3,12,21] with a more recent incidence in one study at 13.0% (n = 574).[36] Pressure injury prevalence ranged from a low of 9% to a high of 60.6% in a retrospective study,[37] with a mean of 7.91% in several studies.[9,12,15,37–42] The vast majority were stages 1 and 2. In a 2005 home hospice study, Reifsnyder and Hoplamazian[43] found a 15% to 27% prevalence of pressure injuries in a population with a 72-year mean age in whom the primary diagnosis was cancer. The primary comorbidity was cognitive-related disorders, with dementia being the primary risk factor.

Tippett[9] conducted a cross-sectional study of 383 hospice patients; 35% had skin wounds, of which 50% were pressure injuries. The same author did a case series analysis of 192 consecutive patients referred for wound consultation. The mean age was 82 years, 67% were

female, and the subjects had multiple comorbidities, with dementia being the primary disorder. The researcher found that 40% of the wounds were pressure injuries, with the primary location being the sacrum, followed by the heel, foot, and leg.[9] In both populations, pressure injuries were almost exclusively stages 3 and 4, with concomitant necrosis and gangrene.[9] Tippett[9] concluded that "wounds at the end of life are a problem of tragic proportion for the nearly 1 million hospice patients and millions of other frail, elderly persons living with chronic disease." The 2014 National Pressure Ulcer Advisory Panel (NPUAP) (note the NPUAP is now the NPIAP as of 2019) Unavoidable Consensus Conference was that not all pressure injuries are avoidable and end of life is one of those times.[22,44,45]The 2019 International Pressure Ulcer Prevention and Treatment Guidelines[46] present evidence-based recommendations, many of which are applicable to palliative care individuals. Unfortunately, but understandably so, a paucity of research evidence exists for this population, leaving the vast majority of the recommendations at the "C" level. International guidelines are also available for venous ulcers[47] as well as arterial and diabetic ulcers.[48,49]

Risk Factors for Skin Breakdown and Pressure Ulcer Development

Several risk factors place individuals at the end of life at risk for the development of skin ulcers as well as impaired healing of skin ulcerations. Advancing age is a well-known risk factor. As one ages, skin becomes drier and more fragile and prone to injury; healing is slowed; the protective function of the epidermis is compromised; and the production of collagen decreases.[11,50–53] Dyspnea, anorexia, and cachexia syndrome, for example, can impair sensory perception.[6–8,12,54] The external forces of pressure and shear deform the skin and underlying tissues, and the tolerance of each individual's tissue to this force is variable. Even with appropriate preventive interventions to avoid the formation of pressure injuries (PIs), comorbidities can overwhelm the skin and potentially decrease tissue tolerances.[46,54–56] Many of the known PI risk factors remain nonmodifiable by current treatment.[46,54] There are numerous other risk factors that will be discussed separately.

Impaired Mobility and Repositioning

Any individual at the end of life experiences prolonged periods of inactivity or immobility, which contribute to the occurrence of tissue ischemia from prolonged pressure.[25,37,46,50,57–63] The risk of tissue breakdown increases when the individual is older, has more comorbid conditions, or has pain that decreases movement. Immobility is the state of being limited in movement or unable to move independently. Immobility is the most significant risk factor for PU development,[64,65] although at present, there are no methods to measure immobility necessitating reliance on clinical characteristics (e.g., ambulation, bed/chair bound, history of CVA).[65] Immobility can be identified via assessment using a mobility subscale on a risk assessment tool[66,67] or an assessment of an individual's ability to participate in ADLs related to mobility and activity.[66] Immobility was identified as the most common risk factor on risk assessment scale for PI development (on 49 of 56 risk scales).[68] There was a 100% concordance for immobility as a significant risk factor by two consensus panels.[46,69] Immobility contributes to vascular congestion and deep edema from fluid shifts[70]; muscle strength is reduced by 1% to 1.5%/d of bed rest[71,72]; 14 days of bed rest will decrease RBC mass altering oxygen transport, leading to dyspnea and activity intolerance.[70]

Particularly vulnerable to pressure from inactivity are the heels, sacrum, and elbows. Suspending the heels over a pillow while supporting the entire length of the leg or using heel protectors is helpful in decreasing heel pressure.[46] A general guideline is to ensure that the individual with impaired mobility is repositioned at periodic intervals, often occurring every 2 to 3 hours but individualized to the individual's preferences.[46,73] The frequency of repositioning is influenced by the support surface the individual is lying on.[46] Repositioning is challenging for a patient who is hemodynamically unstable, has a great deal of pain, is nauseous or vomiting, or is unable to lie on one side or on the back.[21,46] Honor the individual's choices after explaining the rationale for these interventions. (See Chapter 11, Pressure Redistribution: Seating, Positioning, and Support Surfaces.)

Friction and Shear

Along with immobility, friction and shear are risk factors for pressure injury development. Shear stress is the “force per unit area exerted parallel to the plane of interest,” and shear strain is the “distortion or deformation of tissue as a result of shear stress.”[74] Friction is the “resistance to motion in a parallel direction relative to the common boundary of two surfaces.”[74] When combined with pressure, these forces damage and distort tissue and can accelerate tissue damage. To protect the buttocks and sacral areas, use a lift sheet or an overhead trapeze.[46,50,75–78] Protect the sacral area or other bony prominences with a transparent film, silicone, foam, or hydrocolloid to minimize friction.[46] While maintaining the head of the bed at the lowest elevation possible (ideally 30 degrees or less) is recommended to minimize friction and shear to the sacrum and buttocks[46,79,80], this may not be consistent with the patient’s condition or goals. Furthermore, even the 30-degree HOB elevation increased interface pressures.[81] Many individuals at the end of life have impaired ventilation and require the head of the bed to be raised greater than 30 degrees, which would be inconsistent with the pressure injury guidelines.[46] A pressure-redistributing mattress overlay or a specialty bed may also be helpful.[46,79] (See Chapter 11, Pressure Redistribution: Seating, Positioning, and Support Surfaces.)

Nutrition and Hydration

As the body systems are shutting down at the end of life, food and fluid requirements generally decrease as well. Individuals at the end of life have a diminished hunger and thirst mechanism, leading to dehydration, decreased oral intake, and impaired metabolism.[78,82] Lessening of oral intake can occur weeks to months before death.[80] Poor hydration impairs skin turgor, leaving tissue vulnerable to new breakdown. A decrease in protein intake leads to protein wasting and malnutrition. Albumin, a component of protein, provides colloid oncotic pressure to hold liquid in the vascular system. When oncotic pressure is decreased, fluid leaves the vascular system and enters interstitial spaces, causing tissue edema, lowered blood pressure, and impaired blood flow. Nutrition and hydration status can be further impaired if a draining wound causes the loss of large amounts of fluids and proteins in the exudate. At the end of life, the swallow reflex is decreased as well, impairing food and fluid intake and leaving the individual vulnerable to aspiration. Helping the family and loved ones to understand this end-of-life process can relieve their anxiety and stress. (See Chapter 10, Nutrition and Wound Care.)

That protein–calorie malnutrition is associated with pressure injury development in individuals at the end of life has been demonstrated in several studies.[3,37,58,59,81–84] In fact, in a large retrospective cohort study of more than 2,400 adult nursing home residents, Horn et al.[58] documented that an unintentional weight loss at any body mass index increased the chance of developing a pressure injury by 147%. Cytokines, stress response proteins, are produced following tissue injury and contribute to the occurrence of malaise, anorexia, and consequent malnutrition, muscle wasting, and decreased albumin synthesis.[85] (See Chapter 10, Nutrition and Wound Care.)

It is important to maintain adequate nutrition and hydration that is compatible with the condition and wishes of the individual. Maintaining adequate nutritional support may not be attainable. Liberalize food choices, offer several small meals and/or snacks, and offer supplements.[46]

Moisture

Moisture is another risk factor that can arise from perspiration, wound exudate, urine, and/or feces.[6,9,13,15,86] Moisture increases the tissue's susceptibility to damage. Incontinence is of particular importance with palliative care patients because of the risk for skin injury and breakdown. When incontinence is present, the primary goals are to prevent and manage skin breakdown, enhance comfort, and control odor. Feces are a chemical irritant to the skin, and their removal adds the element of a mechanical irritant, so gentleness is important. Skin protectants/barriers are also helpful.[46] It may become necessary to utilize urine (catheter) or feces (fecal) containment devices for skin protection.[6,8]

Individuals at end of life generally have lower scores on the Karnofsky Performance Scale (KPS) and the Palliative Performance Scale,[36,37] and the KPS was a significant predictor of pressure injuries in home palliative care patients ($p < 0.001$, KPS < 30).[36]

Palliative Wound Treatment

The goals of palliative care are to prevent wounds if possible, stabilize and aim to close existing wounds, and manage related symptoms to enhance individual comfort, well-being, and quality of life.[4,6] Palliative wound care differs little from those of curative wound care, aside from the goal of healing. Palliative care is the primary focus when it becomes apparent that the wound has failed to progress or when the patient's clinical condition deteriorates to a point at which aggressive measures are no longer appropriate. This can be particularly true in "nursing home residents with flexion contractures, cognitive impairment, and limited quality of life."[87]

While the exact mechanisms that lead to poor wound healing are not well elucidated, it is reasonable to assume that the cause is an interrelation between local and systemic factors.[88] When it becomes apparent that wound healing would not render the patient's quality of life significantly better, wound stabilization and palliation become the focus of care.[4,6,89] Palliative wound care focuses on controlling pain, choosing appropriate dressings, managing infection, and protecting the periwound area.

Maintenance of Skin Integrity

The goal of palliative care is to optimize quality of life by controlling physical symptoms and monitoring the patient's psychosocial needs. Patients at the end of life are particularly at risk for skin breakdown. They experience a failure of the homeostatic mechanisms, including the skin's ability to counter insults such as pressure, friction, and shear. This includes decreased cutaneous perfusion and local tissue hypoxia, which ultimately reduce oxygen and vital nutrient utilization and removal.[90] The immune system also becomes impaired with skin breakdown. Thus, not all skin breakdown and pressure ulcers are avoidable.[55,90] When breakdown occurs, impaired oxygenation slows healing due to decreased hemoglobin levels, impaired gas exchange, and decreased blood pressure.[91]

It is essential to maintain skin integrity to prevent pressure injuries. Maintaining healthy skin requires both comprehensive assessment and care planning. Inspect skin for erythema, especially over bony prominences frequently, and particularly during repositioning and care activities. Nutrition and hydration play a key role in skin health. Appropriately managing other skin conditions (e.g., rashes, incontinence-associated dermatitis) is necessary to maintain the skin's integrity and ability to protect underlying tissues. Preventing and treating skin breakdown go a long way toward meeting palliative care goals. Skin breakdown can be prevented and treated through risk appraisal and assessment, meticulous skin care, positioning, reducing friction and shear, using support surfaces, providing nutrition and hydration, and managing moisture. Careful attention to skin care in the end-of-life patient is exceedingly important, as a feeling of cleanliness can enhance an overall sense of comfort and well-being and can address any odor problems that might be present. A low-pH skin cleanser is useful along with a moisture barrier to minimize the effects of excess moisture. Excess moisture can cause skin maceration, which interferes with the ability of the skin to withstand friction, shear, and pressure, thus making it vulnerable to injury.[79] Therefore, gently cleanse the skin, paying attention to the sacrum, elbows, and heels, which are prone to friction and pressure injury.[75,76] Avoid massage

over a reddened area as it can further damage tissue with already impaired perfusion.[79] Unless contraindicated, a gentle overall body massage is often appreciated in an individual at the end of life. Emollients and skin protectants are helpful in protecting the skin.[46] Skin emollients applied according to manufacturer's directions are helpful in maintaining adequate skin moisture and preventing dryness.[46] When incontinence is present, it is important to minimize its harmful effects with skin barrier products.

Practice Point

End-of-life wound care focuses on:

- pain management
- odor control
- management of exudates
- bleeding control
- self-image
- dignity
- quality of life

Patient and Wound Assessment

It is essential to perform a complete head-to-toe assessment of the end-of-life patient, including physical and psychosocial health and overall quality of life. A thorough assessment will assist the healthcare professional in realistically evaluating the need for preventive interventions as well as efficacy and cost-effectiveness of achieving closure, should there be tissue breakdown. The assessment should establish both the risk for and the presence or absence of skin breakdown, including the presence of a pressure ulcer and the risk for developing additional ulcers, paying attention to likely etiology. The following areas should be included in the patient assessment[46]:

- comorbid health problems
- medications the patient is taking
- risk factors present in the patient
- nutritional status
- diagnostic test results
- psychosocial implications
- environmental resources
- patient/family goals.

The Pressure Sore Risk Assessment Scale for Palliative Care[75] was developed for use in individuals at or near the end of life. Its seven subscales include sensation, mobility, moisture, activity in bed or chair, nutrition and weight change, skin condition, and friction and shear. Scores can range from 7 to 28, with a score of 12 or lower indicating low risk; 13 to 17, medium risk; 18 to 21, high risk; and 22 or over, very high risk. Risk assessment can be done weekly or with significant changes in condition. (See Box 26-1, *Hunters Hill Marie Curie Centre Pressure Ulcer Risk Assessment*.) Risk reassessments are important given the likelihood of the patient's deteriorating condition. As the end of life approaches, assessments may be performed less frequently in order to enhance comfort.[46]

Box 26-1 Hunters Hill Marie Curie Centre Pressure Ulcer Risk Assessment

Patient's Name Patient ID No. Date of Admission Date of First Assessment

					Date	Date	Date	Date
SENSORY PERCEPTION Ability to respond meaningfully to discomfort related to pressure	**1 No impairment** Communicates discomfort clearly	**2 Slightly limited** Responds to verbal commands but cannot always communicate discomfort or has sensory impairment in one or two extremities	**3 Very limited** Responds only to painful stimuli (i.e., moans or is restless or has sensory impairment over half of body surface) (e.g., spinal cord compression)	**4 Severely impaired** Unresponsive due to impaired consciousness or analgesia/ sedation or has sensory impairment over most of body surface				
MOISTURE Degree of skin exposure to moisture/ fecal matter	**1 Rarely moist** Skin rarely moist	**2 Occasionally moist** Skin occasionally moist; extra linen required once per day approximately	**3 Very moist** Skin often but not always moist Linen changed at least once per shift	**4 Constantly moist** Skin constantly moist with perspiration, urine, or lymphorrhea or in contact with fecal matter				
MOBILITY Ability to mobilize when out of bed	**1 Walks frequently** Walks around bed at least once every 2 h and outside room at least twice a day	**2 Walks occasionally** Walks occasionally during day for short distances but may require assistance	**3 Chairfast** Ability to walk severely limited Must be assisted into chair or wheelchair Spends more than 16 h in chair or bed	**4 Completely immobile** Spends all day in bed or chair—in excess of 20 h (e.g., due to unconsciousness, pain, dyspnea, fatigue)				
ACTIVITY Ability to change body and limb position when in bed or chair	**1 No limitation** Able to change position frequently and unaided	**2 Slightly limited** Makes slight but frequent changes in body or extremity position	**3 Very limited** Only able to make occasional slight changes in body or limb position—usually requires assistance	**4 Immobile** Unable to change body or limb position due to pain, sedation dyspnea, edema, conscious level, etc.				
SKIN CONDITION Observed condition of skin in areas exposed to pressure	**1 Skin condition good** Skin appearance good; no evidence of edema, discoloration, etc.	**2 Fragile skin** Skin thin, fragile, dry, flaky, or edematous (e.g., due to age, steroids, edema, inflammation, or lymphedema)	**3 Skin marks easily** Skin easily marked by support surface	**4 Skin integrity broken** Skin surface altered (e.g., due to incontinence dermatitis, pressure damage, wound, or skin condition)				
NUTR/T/ON/WE/GHT Food intake or weight change pattern	**1 Satisfactory** Food intake very good OR no significant weight change in last 6 mo	**2 Marginally adequate** Weight appears normal. Food intake slightly restricted	**3 Probably inadequate** Significantly underweight or overweight OR poor food intake	**4 Nutritional status very poor/ severe cachexia** Nutritional status unsatisfactory due to cachexia, obesity, or minimal intake				
FRICTION/SHEAR Presence of friction/ shear	**1 No apparent friction/shear** Can lift body or limb completely without sliding when moving in bed or chair	**2 Occasional friction/shear** Occasionally slides down bed or chair or drags body or limbs—due to position and poor muscle strength or fatigue	**3 Frequent sliding** Frequently slides down in bed or chair Patient unable to lift limb or body without dragging (e.g., due to weakness)	**4 Almost constant friction/shear** Continually slides down bed or chair OR severe lymphedema, spasticity, or agitation results in almost constant friction.				
Low: 12 and under; Medium: 13 to 17; High: 18 to 21; Very High: 22 and over				TOTAL SCORE				
				SURFACE				

Adapted with permission from Chaplin, J. "Pressure Sore Risk Assessment in Palliative Care." *Journal of Tissue Viability* 10(1): 27-31, 2000. Used with permission. Copyright © 2000 Tissue Viability Society. Published by Elsevier Ltd. All rights reserved.

Wounds or ulcers in end-of-life patients are often chronic in nature, as healing is significantly impaired due to physical condition and existing comorbidities. A chronic pressure injury has a well-defined border with surrounding nonblanchable erythema. When induration is present, it can extend outward from the wound edges. Many chronic wounds have rolled-under edges that impede wound healing and closure. Wound edges roll under when the wound bed is dry. In response, the wound attempts to preserve what little moisture is present and epithelialization is slowed, leaving the wound bed unepithelialized. Most wounds have drainage. Chronic wound drainage contains destructive enzymes as well as fibroblasts that are less effective at producing collagen to heal the wound.[92] Assess the wound initially and

with each dressing change, but at a minimum weekly unless the individual is actively dying.[46] (See Chapter 5, Acute and Chronic Wound Healing.)

Evidence-Based PracticeWound and Patient Assessment in Palliative Care

Set treatment goals consistent with the values and goals of the individual, with input from the individual's informal caregivers, and develop a treatment plan that supports these values and goals.

Assess the pressure injury initially and reassess at least weekly to monitor progress toward healing.

Good Practice Statements from the European Pressure Ulcer Advisory Panel, National Pressure Injury Advisory Panel, Pan Pacific Pressure Injury Alliance. "Prevention and Treatment of Pressure Ulcers/Injuries: Clinical Practice Guideline". In E. Haesler, ed. The International Guideline. EPUAP/NPIAP/PPPIA, 2019.

Evidence-Based PracticeMaintaining Skin Integrity in Palliative Care Patients

Conduct a comprehensive skin and tissue assessment for all individuals at risk of pressure injuries:

- as soon as possible after admission/transfer to the healthcare service
- as part of every risk assessment
- periodically as indicated by the individual's degree of pressure injury risk
- prior to discharge from the care service.

Good Practice Statements from the European Pressure Ulcer Advisory Panel, National Pressure Injury Advisory Panel, Pan Pacific Pressure Injury Alliance. "Prevention and Treatment of Pressure Ulcers/Injuries: Clinical Practice Guideline". In E. Haesler, ed. The International Guideline. EPUAP/NPIAP/PPPIA, 2019.

Support Surfaces

Support surfaces are “specialized devices for pressure redistribution designed for management of tissue loads, micro-climate, and/or other therapeutic functions (i.e., any mattress, integrated bed system, mattress replacement, overlay, or seat cushion, or seat cushion overlay).”[74] Some support surfaces provide the additional capability of turning or rotation assistance. It is important to match the support surface to the needs of the individual such that the surface redistributes weight over a larger area, thereby minimizing tissue pressures, particularly over bony prominences.[74] These surfaces are helpful for both the bed and the chair. Refer to the EPUAP/NPIAP/PPPIA guideline[46] for more specific recommendations regarding pressure redistribution in palliative care patients.

Practice Point

Individualize the patient’s turning and positioning schedule based on his or her pain tolerance and comfort level.

Evidence-Based PracticePressure Redistribution in Palliative Care Patients

Reposition all individuals with or at risk of pressure injuries on an individualized schedule, unless contraindicated. (Strength of Evidence = B1)

Determine repositioning frequency with consideration to the individual's:

- Skin and tissue tolerance
- General medical condition
- Overall treatment objectives
- Comfort and pain

Good Practice Statement from 2019 Guideline European Pressure Ulcer Advisory Panel, National Pressure Injury Advisory Panel, Pan Pacific Pressure Injury Alliance. "Prevention and Treatment of Pressure Ulcers/Injuries: Clinical Practice Guideline". In E. Haesler, ed. The International Guideline. EPUAP/NPIAP/PPPIA, 2019.

Moore Z, Cowan S. Repositioning for Treating Pressure Ulcers. *Cochrane Database Systematic Review* 1:CD006898, 2015.

Evidence-Based PracticeNutrition and Hydration in Palliative Care Patients

Develop and implement an individualized nutrition care plan for individuals with, or at risk of, a pressure injury who are malnourished or who are at risk of malnutrition. (Strength of Evidence = B2)

European Pressure Ulcer Advisory Panel, National Pressure Injury Advisory Panel, Pan Pacific Pressure Injury Alliance. "Prevention and Treatment of Pressure Ulcers/Injuries: Clinical Practice Guideline". In E. Haesler, ed. The International Guideline. EPUAP/NPIAP/PPPIA, 2019.

Pain Management

Both prevention and treatment of skin breakdown can be uncomfortable for individuals at the end of life. The majority of patients with a pressure injury experience moderate to severe pain,[93–96] especially during dressing changes and wound bed treatments, and the pain can be acute or chronic.[96,97] In fact, 21 of 23 patients (91%) in one study reported a pressure injury as painful.[98] Likewise, in a systematic review of 15 studies addressing the impact of pressure injury pain, the authors concluded that "pain was the most significant consequence of having a pressure injury and affected every aspect of patients' lives."[80] In fact, pain from a pressure injury can be the most distressing symptom an individual might report.[80] It is important to keep in mind that "pain is anything the patient says it is."[99]

Wound pain can be caused by the tumor pressing on nerves and blood vessels or by exposure of the dermis.[100] Pain in wounds often arises from painful procedures, including cleansing and dressing removal, particularly if the dressing is dry and adherent.[101] Given that an individual at or near the end of life has a wound that likely will not heal, the wound and wound pain are chronic in nature. Chronic pain is pain that is persistent and can occur even when the wound is not being manipulated.[102] Concomitant acute pain can occur with dressing changes, treatments, and additional trauma to the area.

One of the most significant goals in palliative care is to alleviate, or at the very least manage, pain. It is therefore essential to perform regular pain assessments.[89,103] Pain assessment tools reported to be valid and reliable include the visual analog scale for pain (VAS pain); the visual rating scale (VRS); the numeric rating scale; the FACES rating scale (FRS); the FLACC scale (face, legs, activity, cry, consolability); and the McGill pain questionnaire (MPQ). A 2005 study[104] identified the VAS pain, the FRS, and the MPQ as valid and reliable to identify pressure injury pain.

A mild to moderate opioid, such as 1 mg morphine or diamorphine mixed with 1 mg of a hydrogel,[105,106] or nonopioid medication can be used to relieve wound pain, as can a topical agent that contains a local

anesthetic (e.g., one with lidocaine, such as EMLA cream, Lidoderm, or Regenecare)[106–108] or a foam dressing containing ibuprofen (not available everywhere).[109,110] One hospital solely dedicated to caring for palliative patients has developed a unique mixture of Balmex and lidocaine 2.75%, which may be applied topically to painful and odorous wounds. They have reported success with this strategy.[111] Wound treatment pain can also be minimized by using minimal mechanical force for cleansing (4 to 15 psi irrigation force); using warmed products, such as normal saline or gauze pads[112]; and avoiding antiseptic and cytotoxic agents.[46,79] Nonpharmacological strategies such as music, relaxation, position changes, meditation, guided imagery, healing touch, distraction, etc. are also available.

Practice Point

Premedicating the patient with pain medication 30 to 60 minutes before changing the dressing is an important part of palliative wound care management.

Wound Dressings

When possible, select a dressing that can remain in place for several days; however, this isn't always possible when a large amount of exudate is present. A dressing that protects periwound skin is also desirable, as is one that protects the wound from moisture and feces. As a rule, nonadherent, foam, polymeric membrane, and/or silicone dressings are best. Maintain a moist wound care environment to prevent exposure of delicate nerve endings,[113] as dry, desiccated wound beds and dressings are nearly always painful.[114] (See Wound Treatment Options, Chapter 9.)

Evidence-Based PracticeTreating Open Pressure Injuries in Palliative Care Patients

Diamorphine HDG is an effective treatment for open pressure injuries (wounds) in the palliative care setting.

Brown, G. "Long-term Outcomes of Full-thickness Pressure Ulcers: Healing and Mortality." *Ostomy/Wound Management* 49:42-50, 2003.
European Pressure Ulcer Advisory Panel, National Pressure Injury Advisory Panel, Pan Pacific Pressure Injury Alliance. "Prevention and Treatment of Pressure Ulcers/Injuries: Clinical Practice Guideline". In E. Haesler, ed. The International Guideline. EPUAP/NPIAP/PPPIA, 2019.

Wound Infection, Odor, and Exudate

Tissue deprived of oxygen and nutrients becomes devitalized and nonviable,[115] and bacteria thrive on this moist, devitalized tissue.[116] As the bacteria colonize, necrotic material appears in the wound and creates an odor, which varies depending on the bacteria present. Nonviable tissue eventually serves as a culture medium to support bacterial growth and inhibit leukocyte phagocytosis of bacteria.[115] Odor is generally associated with anaerobic and some gram-negative (e.g., *Pseudomonas*) microorganisms.[91,117] Anaerobic bacteria are usually present in necrotic material, thrive in the absence of oxygen, and can become buried deeper within the wound. Anaerobic bacteria[114] also have a stronger, more offensive odor that can be particularly distressing to the patient. The wound appearance becomes black and leathery with exposure to air or yellow-gray when exposed to moisture, which occurs over varying lengths of time depending on the underlying disorder.[114]

It is the odor, drainage, and pain arising from the infection that are often the most distressing to the individual and therefore should be treated. Wound odor can be embarrassing to the individual and lead to isolation, depression, and poor quality of life.[2,23,117] Treatment is aimed at removing the cause of the odor and the odor itself. Because saturated dressings can hold odor, more frequent changing may help control the odor as well as the pain from the weight of the dressing. A secondary dressing may help. Frequent irrigation also helps remove exudate and odor. Topical metronidazole gel (0.75% to 0.80%) can be used directly on a wound daily for 5 to 7 days or more often if necessary and tablets can be crushed and placed on the wound bed.[6] Removal of nonviable tissue can be helpful, as it inhibits healing, supports bacterial growth, and can mask signs and symptoms of infection.[4] Nonviable tissue can be débrided, and autolytic débridement is often the least painful for the individual. Sharp débridement is more or less a last resort, and caution must be exercised to prevent excessive bleeding and/or pain.[117,118] Topical metronidazole (gel, cream, crushed tablets, oral) has been shown to be effective for anaerobic bacteria as has silver sulfadiazine.[4,6,46,119–

[127] Activated charcoal dressings have also been shown to be helpful.[88,92,118,123,127–131]

Nonsurgical (autolytic or enzymatic) débridement is recommended due to the tendency for bleeding and "seeding" of malignant cells in fungating and radiation wounds.[114] Topical metronidazole has also been used successfully to control odor.[2,86] Activated charcoal dressings are effective in controlling odor quickly,[23,118,120,124,129,132,133] as are occlusive dressings and frequent dressing changes.[23] Cadexomer iodine is an effective antiseptic,[124] as is povidone–iodine.[126] Silver dressings are effective for treating infections and thus in controlling odor. Dakin's solution (0.25% sodium hypochlorite) is another effective odor controller; it is saturated into gauze and placed in the wound for a limited time and may cause some discomfort.[126] Placement of larvae is another effective method of eliminating infection and controlling odor from wounds with extensive necrotic tissue.[127] Room deodorizers are also helpful. Sugar paste and honey are once again being used for their antibacterial and débriding properties.[130,131] The high sugar content produces a hyperosmotic wound environment to inhibit bacterial growth and assist in débridement.[10,130,131]

Periwound skin protection is crucial because exudate, which is liquid and sometimes caustic, can exacerbate skin damage[133] by causing maceration, breakdown, and itching.[46] Dressings that appropriately control exudate without unnecessarily increasing wetness or dryness are recommended, such as an alginate, Hydrofiber, foam, or a nonadherent dressing (silicone, polymeric foam, foam), often with a secondary absorbent pad.[114,133,134] Be sure to change dressings when they become saturated, as heavy or overly saturated dressings can cause wound bed pain and irritate periwound skin. Alternatively, if exudate is minimal, a low-absorbency dressing, such as a hydrocolloid or semipermeable film, is recommended.[135] A barrier film around the periwound area is helpful in controlling damage from moisture. See Chapter 9, Wound Treatment Options, for further information on dressings.

Evidence-Based PracticeWound Dressing and Odor Control Options for Palliative Care Patients

For all pressure injuries, select the most appropriate wound dressing based on goals and self-care abilities of the individual and/or their informal caregiver and based on clinical assessment including:

- Diameter, shape, and depth of the pressure injury
- Need to address bacterial bioburden
- Ability to keep the wound bed moist
- Nature and volume of wound exudate
- Condition of the tissue in the wound bed
- Condition of the periwound skin
- Presence of tunneling and/or undermining
- Pain

Good Practice Statement from the European Pressure Ulcer Advisory Panel, National Pressure Injury Advisory Panel, Pan Pacific Pressure Injury Alliance. “Prevention and Treatment of Pressure Ulcers/Injuries: Clinical Practice Guideline”. In E. Haesler, ed. The International Guideline. EPUAP/NPIAP/PPPIA, 2019.

Practice Point

Odor control is vital for enhancing quality of life for wound patients receiving palliative care.

The inflammatory fluid that seeps from the extracellular spaces is what is known as exudate. All bacteria produce exudate, the color and odor of which vary according to the causative organism.[114] For example, green exudate generally indicates gram-negative, aerobic bacteria, which respond well to silver found in many dressings now on the market.[114] The more persistent the inflammation or infection is, the more the exudate that will be produced. Exudate frequently contains proteins; when combined with inadequate oral intake of protein commonly seen in the end-of-life individual, the degree of hypoproteinemia can increase. Managing exudates can be a major challenge.[136–142]

Fungating Wounds

Fungating wounds occur when the skin and its supporting blood and lymph vessels are infiltrated by a local tumor or by metastatic spread from a primary tumor, resulting in oxygen starvation to the tissue and eventual necrosis.[124,129,143] It's reported that approximately 5% to 19% of patients with metastatic cancer will develop a fungating wound.[12,144] The incidence in elderly individuals over age 70 is higher.[134,145] Although these wounds often develop during the last months of life, they can be present for years.[23] The most common site for development of a fungating wound is the breast, but they can also be found on the head and neck as well as in an area of melanoma. The anatomical location and the delicacy of the surrounding tissue make it challenging to address these wounds.

The term fungating refers to a malignant process of both ulcerating and proliferative growth through direct invasion.[146–149] An ulcerating wound will produce a craterlike wound, whereas a lesion with a predominantly proliferative growth pattern often develops into a nodular "fungus"- or "cauliflower"-appearing lesion.[144,146] Mixed-appearing lesions can also develop.[144,146] Skin tumors tend to become ulcerated because the skin is a bacterially contaminated surface.[147] Common symptoms of fungating wounds include exudate, pain, odor, pruritus, and bleeding as well as psychosocial issues.[129,143]

Practice Point

Families, support persons, and caregivers may need emotional support when viewing patients with fungating wounds.

Fungating wounds rarely heal[146]; thus, management is centered on symptom control, promotion of comfort, and maintenance or

improvement of quality of life.[144,148] Assessment and management by healthcare providers, especially the nurse and physician, are most challenging.[144] Therefore, excellent interdisciplinary care and ongoing patient–caregiver communication are essential.

Care of Fungating Wounds

Woo and Sibbald[88] proposed an acronym for treatment of fungating wounds. HOPES provides a systematized approach and includes **H**emorrhage, **O**dor, **P**ain, **E**xudate, and **S**uperficial infection.[88] Hemorrhage due to erosion of blood vessels is the most common emergency seen in fungating wounds and can also be related to the decreased platelet function within the tumor.[149] Blood vessels can become eroded from the tumor cells themselves or secondary to necrosis or sloughing of tissues after radiotherapy.[150] Nonsurgical (autolytic or enzymatic) débridement is recommended due to the tendency for bleeding and "seeding" of malignant cells.[114,133] Fungating lesions are friable and predisposed to bleeding. To minimize bleeding, use a nonadherent or soft silicone dressing, maintain a moist wound bed, and clean by gentle irrigation rather than swabbing.[147] Dry dressings can cause bleeding when they adhere to the wound bed and should be avoided.[114] Alginate dressings have a high seaweed content and exchange sodium ions for calcium ions in the wound bed, thus encouraging the clotting cascade. Alginates must be used with caution in fragile tumors, however, because these dressings can also cause bleeding.[105] Other natural hemostats are collagen and oxidized cellulose; silver nitrate is a sclerosing agent; epinephrine is a vasoconstrictor; and sucralfate is an astringent.[114,129,145] Hemostatic surgical sponges can also be used and left in place for a time.[23] Alginates, foam, silicone, and polymeric foam dressings are helpful for odor.[151,152]

Radiation Wounds

Radiation therapy targets a high-energy x-ray beam to an area of treatment. The target area is usually a tumor, the area surrounding a tumor, or an area where a tumor has been surgically removed. While each treatment is designed to target tissue at a particular depth, the tissues overlying the site can be affected as well.[146,153]

Radiation-related skin changes or ulcerations can occur in soft tissues during the course of therapy, immediately after therapy, or a long time following therapy.[154,155] Skin problems can also be noted in individuals who underwent treatments years ago before technological improvements in radiation machines were implemented. The skin reactions seen are generally specific to the area that was irradiated, and the inflammation can occur almost immediately.[155,156] Acute erythematous wounds result from the dilated blood vessels in the irradiated area. The ulceration may be large and may present initially as a draining sinus.[157]

The more common skin reactions associated with radiation therapy include flaking or peeling, redness, changes in pigmentation, loss of hair, decreased or absent perspiration, superficial blood vessel changes, edema, ulceration, and scarring.[147,155] (See Box 26-2, *Common radiation wound skin reactions*.)

Box 26-2 Common Radiation Wound Skin Reactions

- Flaking or peeling (dry desquamation)
- Erythema
- Alteration in pigmentation
- Hair loss
- Loss of perspiration or sebaceous excretion
- Changes in superficial blood vessels
- Edema
- Ulceration (moist desquamation)
- Scarring

From Smith, S. *Skin Care Following Radiation Therapy: The Clinician's Notebook*. Irving, TX: Carrington Laboratories, Inc. Newsletter; 1(3):1-3. Retrieved from

http://www.woundcare.org/newsvol2n2./ar3.htm. Last accessed January 12, 2019.

Changes at the cellular level can be reflected by poor healing at the site. Healing is impeded related to atrophy of the epidermis and epidermal accessory structures, microvascular occlusions, exuberant connective tissue, decreased fibroblast reproduction, and significant amounts of cellular damage.[147,157]

Most radiation-related lesions are superficial. In 1994, the Oncology Nursing Society created a classification system for radiation ulcers. This system was refined in 2002. The five-level classification system ranges from “0” or no skin problem within a radiation field to “4” or skin necrosis or ulceration of full-thickness dermis[158] (Table 26-1).

Table 26-1 Oncology Nursing Society Classification for Skin Reactions

0	None
1	Faint erythema or dry desquamation
2	Moderate to brisk erythema or patchy moist desquamation, mostly confined to skin folds and creases; or moderate edema
3	Confluent moist desquamation ≥1.5 cm in diameter and not confined to skin folds; pitting edema
4	Skin necrosis or ulceration of full-thickness dermis; may include bleeding not induced by minor trauma or abrasion

Adapted from Oncology Nursing Society. *Radiation Therapy Patient Care Record: A Tool for Documenting Nursing Care*. Pittsburgh, PA: Author, 2002, with permission of the publisher.

Treatment of a radiation-induced skin lesion is essentially like treatment for other types of wounds. Any tissue within a radiation field must be considered at high risk for potential breakdown and should be kept clean, appropriately moistened, and protected from potential injury. Skin can also be protected by avoiding restrictive clothing, adhesives, harsh chemicals, heat or sunlight, and trauma. Should a minor skin reaction such as erythema or dry desquamation occur, the same guidelines apply, along with use of a topical hydrogel or a steroid cream.[147] Moist desquamation is also treated in the same manner as described above, with the addition of a nonadherent or foam dressing to manage the wound environment. It's important to cover the wound to prevent evaporation of fluid, control pain, and reduce risk of infection.[155,156]

Severe ulceration or necrosis needs to be treated as an open wound, using moist wound healing principles.[147,155] However, it's important to first rule out a new malignancy in the area.[156] Skin grafting or growth factor application may be required.[143] As a consequence of the vascular changes and resultant hypoxia, irradiated tissues have a decreased ability to fight infection. Avoiding or controlling infection is important, and antibiotics are best delivered topically.[157] The vascular changes and hypoxia also are responsible for pain being present in these ulcerations.[157] These wounds are typically difficult to manage and slow to heal. In all instances, systemic support is necessary to enhance the patient's healing potential in order to minimize further trauma to the wound site.[155]

Summary

While cure and/or healing of a wound is not always realistic, it's possible to provide compassionate and symptom-relieving treatment for palliative care patients who have wounds. This includes balancing the management of local wound symptoms, such as pain, odor, exudate, and bleeding, while preserving patient dignity and self-esteem and maximizing quality of life. Few randomized clinical trials or other research studies exist in the area of palliative wound care. However, there is a consensus document from the International Palliative Wound Care Initiative that looks at managing these wounds across the life continuum.[159] Continued study is needed to more clearly understand when a palliative care goal is appropriate. A comprehensive palliative wound care program needs to be developed in clinical agencies that work with these patients. The interdisciplinary team would include the physician, nurses, wound care specialists, dietitian, chaplain services, social services, and pain and hospice consultants.[76] Palliative care units are increasing in number, particularly for patients who are chronically, but not terminally, ill.[160] Wounds treated appropriately, even when the goal is not healing, can markedly improve in 50% of the cases, even on a hospice unit.[8,161,162]

PATIENT SCENARIO

Clinical Data

Mr. M is an 86-year-old widower who has end-stage cancer with bone metastasis. Because he is quite coherent, refuses to be hospitalized, and insists on staying in his home, his family is caring for him with the assistance of hospice. While Mr. M is consuming liquids fairly well, his food intake is very minimal; he eats very little, even with encouragement. With his small frame, his current weight is 109 lbs, down from his usual weight of about 165 lbs. He remains continent and uses a bedside commode with assistance. Upon assessment, the hospice nurse identifies a stage 3 pressure injury over the left greater trochanter measuring 2.5 × 2.3 × 0.3 cm, and there is a moderate

amount of tan/green exudate with a slightly foul odor emanating from it. He also has a stage 4 medical device–related pressure injury measuring 0.25 × 0.25 cm on the left ear.

The family has tried both a sheepskin and an air overlay on his bed, but Mr. M insisted they be removed because he didn't like them. Mr. M informs the hospice nurse that he is "ready to go, and doesn't wish for any fancy or heroic treatments. Just let me be."

Case Discussion

Based on the initial assessment, the hospice nurse identified a number of problems that needed to be addressed. The assessment included ascertaining both Mr. M's goals of care and those of his family. Mr. M expressed his desire to just be kept comfortable. His wish was to remain in his own bed with a "pillow top" mattress rather than in a "hospital" bed, and he was adamant that he did not wish to have any other pressure-redistributing devices on his bed. While a plan of care would include regular repositioning, Mr. M verbalized that "it hurt less to lie on my left side on the pressure injury than in any other position." The nurse educated him on the risk for further breakdown of the PI and ear as well as other areas of his skin. With encouragement from his family, Mr. M agreed to lie on his back and right side for 30 minutes each and on his left side for 2 hours at a time, on a rotating basis. Bilateral heel protectors were used, and his legs were elevated with pillows placed lengthwise in the bed.

Mr. M's PI was cleansed at each dressing change with an antiseptic solution to assist with odor management and covered with a composite dressing. A foam dressing was used on the ear to cushion and protect it. A pie tin with charcoal was placed under his bed to absorb room odor. A skin protectant was used, particularly over bony prominences, to help prevent further breakdown and protect his fragile skin.

Mr. M's dietary likes and dislikes were also assessed. After being educated on the need for protein for strength and energy to get up to the bedside commode, he agreed to consume three high-protein liquid drinks per day, along with popsicles, ice cream, and occasionally oatmeal and a piece of cold meat rolled with a slice of cheese, as tolerated.

After pain assessment as well as 30 minutes prior to dressing changes, Mr. M was medicated using a nonopioid medication. Mr. M was encouraged to request a “time-out” during dressing changes if needed.

While the pressure injury over the left greater trochanter did not heal, it only increased in size to 3.5 × 3.5 × 0.3 cm prior to his death. This outcome was within the goal of the patient and family. The injury on the left ear did not heal either, nor did it increase in size. Taking care to position Mr. M’s head when he was on his left side proved helpful in preventing deterioration. Mr. M remained coherent up until his death. Two days prior to his death, Mr. M expressed his appreciation for allowing him to do things “his way.”

Show What You Know

1. **Which of the following defines palliative care?**
   A. An organized and highly structured system to deliver care focused on promoting the greatest comfort and dignity of the patient
   B. Care that affirms life yet strives to deliver highly organized care to an individual who is focused on regaining a former health state
   C. A care delivery system focused on wound healing and elimination of symptoms
   D. Care that is delivered at home by loved ones without the involvement of healthcare providers

2. **Which of the following situations would constitute palliative wound care?**
   A. Wet-to-dry dressing changes every 4 hours around the clock
   B. Calcium alginate dressings used on a necrotic-appearing wound with minimal exudate
   C. Silver-impregnated dressings used on a wound with little evidence of inflammation and essentially no evidence of infection
   D. A hydrogel dressing placed on a wound every 3 days and as needed

3. **Which of the following orders should the clinician question in caring for a fungating wound?**
   A. Using nonadherent dressings
   B. Using cold saline when irrigating the wound
   C. Using a mixture of morphine with amorphous hydrogel
   D. Using music and other relaxation techniques when providing care

4. **A patient with a grade 3 (Oncology Nursing Society classification system) skin reaction would require which the**

following interventions?

A. None, skin is normal
B. Frequent application of skin moisturizer
C. Use of a protective skin barrier
D. Enzymatic débridement ointment three times per day

Answers to these questions are found on page 688.

References

1. World Health Organization. Retrieved from http://www.WHO.int/cancer/palliative/definition/en/. Last accessed June 12, 2014.
2. National Institutes of Health. Improving End-of-Life Care. State-of-the-Science Conference Statement. Bethesda, MD: Author, December 6-8, 2004.
3. Alvarez, O., et al. “Chronic Wounds: Palliative Management for the Frail Population.” Wounds 14(8 Suppl): 1-27, 2002.
4. Nenna, M. “Pressure Ulcers at End of Life: An Overview for Home Care and Hospice Clinicians.” Home Healthcare Nurse 2011;29(6):350-65.
5. Temple K. Palliative Care. The Rural Health Monitor, 2018.
6. Langemo, D.K., Black, J. “Pressure Ulcers in Individuals Receiving Palliative Care: A National Pressure Ulcer Advisory Panel White Paper.” Advances in Skin & Wound Care 23(2):59-72, 2010.
7. Walding, M., Andrews, C. “Preventing and Managing Pressure Sores in Palliative Care.” Professional Nurse 11:33-8, 1995.
8. Langemo, D.K., Brown, G. “Skin Fails Too: Acute, Chronic, and End-Stage Skin Failure.” Advances in Skin & Wound Care 19(4):206-11, 2006.
9. Tippett, A.W. “Wounds at the End of Life.” Wounds 17(4):91-8, 2005.
10. Naylor, W. “Malignant Wounds: Aetiology and Principles of Management.” Nursing Standard 16: 45-56, 2002.
11. Henoch, I., Gustafsson, M. “Pressure Ulcers in Palliative Care: Development of a Hospice Pressure Ulcer Risk Assessment Scale.” International Journal of Palliative Nursing 9:474-84, 2003.
12. Maida, V., Corbo, M., Dolzhykov, M., et al. “Wounds in Advanced Illness: A Prevalence and Incidence Study Based on a Prospective Case Series.” International Wound Journal 5:305-14, 2008.
13. Maida, V., Ennis, M., Corban, J. “Wound Outcomes in Patients with Advanced Illness.” International Wound Journal 9(6):683-92, 2012.
14. Aminoff, B.Z., Adunsky, A. “Dying Dementia Patients: Too Much Suffering, Too Little Palliation.” American Journal of Alzheimer’s Disease and Other Dementias 19:243-7, 2004.
15. Hendrichova, I., Castelli, M., Mastroianni, C, et al. “Pressure Ulcers in Cancer Palliative Care Patients.” Palliative Medicine 24:669-73, 2010.
16. Froiland, K.G. “Wound Care of the Advanced Cancer Patient.” Hematology/Oncology Clinics of North America 16:629-39, 2002.
17. Langemo, D.K., Anderson, J., Hanson, D., et al. “Understanding Palliative Wound Care.” Nursing 37: 65-6, 2009.
18. Tippett, A.W. “Wounds at the End of Life.” Journal of Palliative Medicine 8(1):243, 2005.
19. Baharestani, M.M. “The Lived Experience of Wives Caring for Their Frail, Home Bound, Elderly Husbands with Pressure Ulcers.” Advances in Skin &

Wound Care 7:40-52, 1994.
20. Bale, S., et al. “Pressure Sore Prevalence in a Hospice.” Journal of Wound Care 4(10):465-6, 1995.
21. Hatcliffe, S., Dawe, R. “Monitoring Pressure Sores in a Palliative Care Setting.” International Journal of Palliative Nursing 2(4):182-6, 1995.
22. National Pressure Ulcer Advisory Panel. “Avoidable Versus Unavoidable Pressure Ulcers.” Journal of Wound, Ostomy, and Continence Nursing 44(5):458-68, 2017.
23. Naylor, W. World Wide Wounds: Part 1: Symptom Control in the Management of Fungating Wounds. Retrieved from http://www.worldwidewounds.com/2002/march/Naylor/Symptom-Control-Fungating-Wounds.html. Last accessed May 19, 2014.
24. Bennett, R.G., et al. “Medical Malpractice List Related to Pressure Ulcers in the United States.” Journal of the American Geriatrics Society 48:73-81, 2000.
25. DeConno, F., Ventafridda, V., Saita, L. “Skin Problems in Advanced and Terminal Cancer Patient.” Journal of Pain and Symptom Management 6:247-56, 1991.
26. Gilchrist, B., Corner, J. “Pressure Sores: Prevention and Management—A Nursing Perspective.” Palliative Medicine 3:257-61, 1989.
27. Moss, R.J., LaPuma, J. “The Ethics of Pressure Sore Prevention and Treatment in the Elderly: A Practical Approach.” Journal of the American Geriatrics Society 39:905-8, 1991.
28. Waller, A., Caroline, N.L. “Pressure Sores.” In Handbook of Palliative Care in Cancer. Boston, MA: Butterworth Heinemann, 2001:91-8.
29. Weissman, D.E. End-of-Life Cares Eases Pain and Prepares Patient for Death. Health Link, Medical College of Wisconsin, 2003. Retrieved from http://healthlink.mcw.edu/article/100171698.html. Last accessed 25 June 2014.
30. Singer, P.A., Bowman, K. “Quality End-of-Life Care: A Global Perspective.” BMS Palliative Care 1:4, 2002.
31. Gottrup F. A Specialized Wound-Healing Center Concept: Importance of a Multidisciplinary Department Structure and Surgical Treatment Facilities in the Treatment of Chronic Wounds. American Journal of Surgery 2004;187(5):S38-43.
32. Rice, K.N., et al. “Factors Influencing Models of End-of-Life Care in Nursing Homes: Results of a Survey of Nursing Home Administrators.” Journal of Palliative Medicine 7(5):668-75, 2004.
33. Covinsky, K.E., et al. “The Last 2 Years of Life: Functional Trajectories of Frail Older People.” Journal of the American Geriatrics Society 51(4):492-8, 2003.
34. Schim, S.M., Cullen, B. “Wound Care at End of Life.” Nursing Clinics of North America 40(2):281-94, 2005.
35. Langemo, D.K. “Pressure Ulcers in Individual at the End of Life: Palliative and Hospice Care.” In B. Pieper, ed. Pressure Ulcers: Prevalence, Incidence and Implications for the Future. Washington, DC: NPUAP, 2012.

36. Artico M, Dante A, D'Angelo, D., et al. Prevalence, Incidence and Associated Factors of Pressure Ulcers in Home Palliative Care Patients: A Retrospective Chart Review. Palliative Medicine 32(1):299-307, 2018.
37. Reifsnyder, J., Magee, H. "Development of Pressure Ulcers in Patients Receiving Home Hospice Care." Wounds 17:74-9, 2005.
38. Galvin, J. "An Audit of Pressure Ulcer Incidence in a Palliative Care Setting." International Journal of Palliative Nursing 8(5):214-21, 2002.
39. Hanson, D., et al. "The Prevalence and Incidence of Pressure Ulcers in the Hospice Setting: Analysis of Two Methodologies." American Journal of Hospice & Palliative Care 8(5):18-22, 1991.
40. Kayser-Jones, J., Kris, A.E., Lim, K., et al. "Pressure Ulcers Among Terminally Ill Nursing Home Residents." Research in Gerontological Nursing 1(1):14-24, 2008.
41. Bonaldi, A., Parazzini, F., Corli, O. et al. "Palliative Care at Home in Milan." European Journal of Palliative Care 16(1):40-2, 2009.
42. Thein, H.H., Gomes, T., Krahn, M.D., et al. "Health Status Utilities and the Impact of Pressure Ulcers in Long-Term Care Residents in Ontario." Quality of Life Research 19(1):81-9, 2010.
43. Reifsnyder, J., Hoplamazian, L. "Incidence and Prevalence of Pressure Ulcers in Hospice." Journal of Palliative Medicine 8(1):244, 2005.
44. Alvarez, O., Brindle, C.T., Langemo, D., et al. "The VCU Pressure Ulcer Summit: The Search for a Clearer Understanding and More Precise Definition of the Unavoidable Pressure Injury." Journal of Wound, Ostomy, and Continence Nursing 43:455-63, 2016.
45. Trombley, K., Brennan, M.R., Thomas, L., et al. Prelude to Death or Practice Failure? Trombley-Brennan Terminal Tissue Injury. American Journal of Hospice and Palliative Medicine 29:541-5, 2012.
46. European Pressure Ulcer Advisory Panel, National Pressure Injury Advisory Panel, Pan Pacific Pressure Injury Alliance. "Prevention and Treatment of Pressure Ulcers/Injuries: Clinical Practice Guideline." In E. Haesler, ed. The International Guideline. EPUAP/NPIAP/PPPIA, 2019.
47. Scottish Intercollegiate Guidelines Network. Management of Chronic Venous Leg Ulcers. A National Guideline. Edinburgh, Scotland: Scottish Intercollegiate Guidelines Network, 2010. Retrieved from www.sign.ac.uk/pdf/sign120.pdf. Last accessed 26 June 2014.
48. Whitney, J., Phillips, L., Aslam, R., et al. "Guidelines for the Treatment of Pressure Ulcers." Wound Repair and Regeneration 14(6):663-9, 2006.
49. Wound Healing Society. Chronic Wound Care Guidelines. Maitland, FL: Wound Healing Society, 2007.
50. Bergquist, S., Frantz, R.A. "Pressure Ulcers in Community-based Older Adults Receiving Home Care: Prevalence, Incidence, and Associated Risk Factors." Advances in Skin & Wound Care 12:339-51, 1999.
51. Ersser, S., et al. "Best Practice in Emollient Therapy: A Statement for Health Care Professionals." Dermatology Nursing 6:S2-19, 2007.

52. Davies, A. “Management of Dry Skin Conditions in Older People.” Nursing Standard 13:250-7, 2008.
53. Lawton, S. “Effective Use of Emollients in Infants and Young People.” Nursing Standard 19:44-50, 2004.
54. White-Chu, E.F., Reddy, M. Pressure Ulcer Prevention in Patients with Advanced Illness. Current Opinion Support. Palliative Care 7:111-3, 2013.
55. Witkowski, J.A., Parish, L.C. “The Decubitus Ulcer: Skin Failure And Destructive Behavior.” International Journal of Dermatology 39:892-8, 2000.
56. Thomas, D.R. “Issues and Dilemmas in the Prevention and Treatment of Pressure Ulcers: A Review.” Journal of Gerontology 56:M328-40, 2001.
57. Brink, P., Smith, T.F., Linkewich, B. “Factors Associated with Pressure Ulcers in Palliative Home Care.” Journal of Palliative Medicine 9:1369-75, 2006.
58. Horn, S.D., et al. “The National Pressure Ulcer Long-term Care Study: Pressure Ulcer Development in Long-Term Care Residents.” Journal of the American Geriatrics Society 52(3):359-67, 2004.
59. Brown, G. “Long-term Outcomes of Full-Thickness Pressure Ulcers: Healing and Mortality.” Ostomy/Wound Management 49:42-50, 2003.
60. Ferrell, B.A., Josephson, K., Norvid, P. “Pressure Ulcers Among Patients Admitted to Home Care.” Journal of the American Geriatrics Society 48:1042-7, 2000.
61. Pang, S.M., Wong, T.K. “Predicting Pressure Sore Risk with the Norton, Braden, and Waterlow Scales in a Hong Kong Rehabilitation Hospital.” Nursing Research 47:147-53, 1998.
62. Perneger, T.V., Gaspoz, J.M., Rae, A.C., et al. “Contribution of Individual Items to the Performance of the Norton Pressure Ulcer Prediction Scale.” Journal of the American Geriatrics Society 46:1282-6, 1998.
63. Salzburg, C.A., Byrne, D., Cayten, C.G., et al. “Predicting and Preventing Pressure Ulcers in Adults with Paralysis.” Advances in Skin & Wound Care 11:237-46, 1998.
64. Cox, J. “Prediction of Pressure Ulcers in Adults Critical Care Patients.” American Journal of Critical Care 20(5): 264-75, 2011.
65. Bours, D., De Laat, E., Halfens, R., et al. “Prevalence, Risk Factors and Prevention Of Pressure Ulcers in Dutch Intensive Care Units.” Intensive Care Medicine 27(10):1599-605, 2001.
66. Bergquist-Beringer, S., Daley, C.M. Adapting Pressure Ulcer Prevention for Use in Home Health Care. Journal of Wound, Ostomy, and Continence Nursing 38(2):145-54, 2011.
67. Bergquist-Beringer, S., Gajewski, B. OASIS Data that Predict Pressure Ulcer Development in Older Adult Home Health Patients. Advances in Skin & Wound Care 2011;24(9):404-14.
68. Garcia-Fernandez, F.P., Agreda, J.J., Verdu, J., et al. “A New Theoretical Model for the Development of Pressure Ulcers and Other Dependence-Related Lesions.” Journal of Nursing Scholarship 46:28-38, 2014.

69. Black, J.M.., Edsberg, L.E., Baharestani, M.M., et al. “Pressure Ulcers: Avoidable or Unavoidable? Results of the National Pressure Ulcer Advisory Panel Consensus Conference.” Ostomy/Wound Management 57(2):24-37, 2011.
70. Winkelman, C. “Bed Rest in Health and Critical Illness: A Body Systems Approach.” AACN Advanced Critical Care 20(3):254-66, 2009.
71. Topp, R., Ditmyer, M., King, K., et al. “The Effect of Bed Rest and Potential Rehabilitation on Patients in the Intensive Care Unit.” AACN Clinical Issues 13(2):263-76, 2002.
72. DeJonghe, B., Bastuji-Garin, S., Durand, M.C., et al. “Respiratory Weakness Is Associated with Limb Weakness and Delayed Weaning in Critical Illness.” Critical Care Medicine 39:2007-15, 2007.
73. Rich, S.E., Margolis, D., Shardell, M., et al. “Frequent Manual Repositioning and Incidence of Pressure Ulcers Among Bed-Bound Elderly Hip Fracture Patients.” Wound Repair and Regeneration 19(1):10-8, 2011.
74. National Pressure Ulcer Advisory Panel. Support Surface Standards Initiative: Terms and Definitions Related to Support Surfaces. Retrieved from http://www.npuap.org/s3i.htm. Last accessed 28 June 2010.
75. Chaplin, J., McGill, M. “Pressure Sore Prevention.” Palliative Care Today 8(3):38-9, 1999.
76. Dealey, C. The Care of Wounds. Oxford, UK: Blackwell, 1999.
77. Peerless, J., et al. “Skin Complications in the Intensive Care Unit.” Clinics in Chest Medicine 20(2):453-67, 1999.
78. Emanuel, L., Ferris, F.D., von Gunten, C.F., et al. “End-of-Life Care in the Setting of Cancer: Withdrawing Nutrition and Hydration.” *EPEC-O: Education in Palliative and End-of-Life Care for Oncology (Module 11: Withdrawing Nutrition, Hydration)*. Chicago, IL: The EPEC Project, 2005.
79. Wound, Ostomy, Continence Nurses Society. Guideline for Prevention and Management of Pressure Ulcers. Glenview, IL: Author, 2003.
80. Gorecki, C., et al. “Impact of Pressure Ulcers on the Quality of Life in Older Patients: A Systematic Review.” Journal of the American Geriatrics Society 57:1175-83, 2009.
81. Bergstrom, N., Braden, B. “A Prospective Study of Pressure Sore Risk Among Institutionalized Elderly.” Journal of the American Geriatrics Society 40(8):747-58, 1992.
82. Thorpe, J. The Importance of Water in Modern Wound Care. Wound Essentials 5:15-118, 2010. Retrieved from www.wounds-uk.com/pdf/content9502pdf. Last accessed 19 September 2019.
83. Berlowitz, D.R., Wilking, S.V. “Risk Factors for Pressure Sores: A Comparison of Cross-Sectional and Cohort-Derived Data.” Journal of the American Geriatrics Society 37:1043-59, 1989.
84. Pinchovsky-Devin, G., Kaminski, M.V. “Correlation of Pressure Sores and Nutritional Status.” Journal of the American Geriatrics Society 34:435-40, 1986.

85. Posthauer, M.E., Thomas, D.R. “Nutrition and Wound Care.” In S. Baranoski, E.A. Ayello, eds. Wound Care Essentials: Practice Principles, 2nd ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2008:197.
86. Grey, J., Enoch, S., Harding, K.G. “Wound Assessment.” British Medical Journal 332:285-8, 2006.
87. Olson, K., et al. “Preventing Pressure Sores in Oncology Patients.” Clinical Nursing Research 7(2):207-24, 1998.
88. Woo, K.Y., Sibbald, R.G. “Local Wound Care for Malignant and Palliative Wounds.” Advances in Skin & Wound Care 23:417-28, 2010.
89. Burt, T. “Palliative Care of Pressure Ulcers in Long-Term Care: Clinical Care and Aging.” Annals of Long-Term Care 21(3):20-8, 2013.
90. Sibbald, R.G., Krasner, D.L., Lutz, J. “SCALE©: Skin Changes at Life’s End: Final Consensus Statement: October 1, 2009.” Advances in Skin & Wound Care 23:225-38, 2010.
91. Sussman, C. “Wound Healing Biology and Chronic Wound Healing.” In C. Sussman, B. Bates-Jensen, eds. Wound Care: A Collaborative Practice Manual for Physical Therapists and Nurses. Gaithersburg, MD: Aspen Publishers, 1998.
92. Maklebust, J., Sieggreen, M. Pressure Ulcers: Guidelines for Prevention and Management, 3rd ed. Philadelphia, PA: Lippincott Williams, & Wilkins, 2001.
93. Fox, C. “Living with a Pressure Ulcer: A Descriptive Study of Patients’ Experiences.” British Journal of Community Nursing 7:10, 12, 14, 16, 20, 22, 2002.
94. Rastinehad, D. “Pressure Ulcer Pain.” Journal of Wound, Ostomy, and Continence Nursing 33:252-6, 2006.
95. Bale, S., et al. “The Experience of Living with a Pressure Ulcer.” Nursing Times 103:42-3, 2007.
96. Dallam, L., et al. “Pain Management and Wounds.” In S. Baranoski, E.A. Ayello, eds. Wound Care Essentials: Practice Principles, 2nd ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2008:229-51.
97. Pasero, C.L. “Procedural Pain Management.” American Journal of Nursing 98(7):18-20, 1998.
98. Spilsbury, K., Nelson, A., Cullum, N., et al. “Pressure Ulcers and Their Treatment and Effects on Quality of Life: Hospital Inpatient Perspective.” Journal of Advanced Nursing 57:494-504, 2007.
99. McCaffrey, M., Pasero, C. Pain: Clinical Manual, 2nd ed. St. Louis, MO: Mosby, 1999.
100. Manning, M.P. “Metastasis to Skin.” Seminars in Oncology Nursing 14(3):240-3, 1998.
101. Jones, M., et al. “Dressing Wounds.” Nursing Standard 12(39):47-52; quiz 55-6, 1998.
102. Krasner, D. “The Chronic Wound Pain Experience: A Conceptual Model.” Ostomy/Wound Management 41(3):20-9, 1995.

103. Langemo, D.K. “When the Goal is Palliative Care.” Advances in Skin & Wound Care 19(3):148, 150-4, 2006.
104. De Laat, E.H., Scholte op Reimer, W.J., van Achterberg, T. “Pressure Ulcers: Diagnostics and Interventions Aimed at Wound-Related Complaints: A Review of the Literature.” Journal of Clinical Nursing 14(4):464-72, 2005.
105. Grocott, P. “Controlling Bleeding in Fragile Fungating Tumors.” Journal of Wound Care 7(7):342, 1998.
106. Twillman, R.K., et al. “Treatment of Painful Skin Ulcers with Topical Opioids.” Journal of Pain and Symptom Management 17(4):39-42, 1997.
107. Smith, N.K., et al. “Non-Drug Measures for Painful Procedures.” American Journal of Nursing 97(8):18-20, 1997.
108. Briggs, M., Nelson, E.A. Topical Agents or Dressings for Pain in Venous Leg Ulcers. Oxford, UK: The Cochrane Library, 2001.
109. Jorgensen, B., Friis, G.I., Gottrup, F. “Pain and Quality of Life for Patients with Venous Leg Ulcers: Proof of Concept of Efficacy of Biatain-Ibu, a New Pain Reducing Wound Dressing.” Wound Repair and Regeneration 14:233-9, 2006.
110. Sibbald, R.G., et al. “A Pilot (Real-life) Randomized Clinical Evaluation of a Pain-relieving Foam Dressing: Ibuprofen-foam versus Local Best Practice.” International Wound Journal 4:16-23, 2007.
111. Kalinski, C., et al. “Effectiveness of Topical Formulation Containing Metronidazole for Wound Odor and Exudates Control.” Wounds 17:74-9, 2005.
112. Hollingworth, H. “Wound Care—Less Pain, More Gain.” Nursing Times 93(46):89-91, 1997.
113. Hallett, A. Fungating Wounds. Wound Care Society Education Leaflet. Huntingdon, UK: Wound Care Society, 1993.
114. Hampton, S. “Managing Symptoms of Fungating Wounds.” Journal of Cancer Nursing 20(1):21-8, 2006.
115. Slavin, J. “Wound Healing: Pathophysiology.” Surgery 17(4):I-IV, 1999.
116. Rodeheaver, G. “Wound Cleaning, Wound Irrigation, Wound Disinfection.” In D.L. Krasner, G.T. Rodeheaver, R.G. Sibbald, et al., eds. Chronic Wound Care: A Clinical Sourcebook for Healthcare Professionals, 4th ed. Wayne, PA: HMP Communications, 2008.
117. McDonald, A., Lesage, P. “Palliative Management of Pressure Ulcers and Malignant Wounds in Patients with Advanced Illness.” Journal of Palliative Medicine 9:285-95, 2006.
118. Eisenberger, A., Zeleznik, J. “Pressure Ulcer Prevention and Treatment in Hospices: A Qualitative Analysis.” Journal of Palliative Care 19:9-14, 2003.
119. European Wound Management Association (EWMA). Position Document: Management of Wound Infection. London, UK: MEP Ltd, 2006:1-6, 11-3.
120. Grocott, P. “Palliative Management of Fungating Malignant Wounds.” Journal of Community Nursing 14(3), 2000. Retrieved from http://www.jcn.co.uk.backiss.asp?YearNum=2000&MonthNum=03&ArticleID=221. Last accessed July 14, 2010.

121. Paul, J.C., Pieper, B.A. “Topical Metronidazole for the Treatment of Wound Odor: A Review of the Literature.” Ostomy/Wound Management 54(3):18-27, 2008.
122. Grocott, P. “Care of Patients with Malignant Fungating Wounds.” Nursing Standard 21(24):57-8, 60, 62, 2007.
123. Young, C.V. “The Effects of Malodorous Fungating Malignant Wound on Body Image and Quality of Life.” Journal of Wound Care 14:359-62, 2005.
124. Williams, C. “Role of CarboFlex in the Nursing Management of Wound Odour.” British Journal of Nursing 10:123-35, 2001.
125. Falanga, V. “Iodine-containing Pharmaceuticals: A Reappraisal.” *Proceedings of the 6th European Conference on Advances in Wound Management*. London, UK: Macmillan Mags Ltd, 1997.
126. Ferris, F., von Gunten, C. Malignant Wounds. 2nd ed. Fast Facts and Concepts #46. End-of-Life Physician Education Resource Center, 2005. Retrieved from http://www.eperc.mcw.edu. Last accessed September 9, 2009.
127. Thomas, S., et al. “Odour Absorbing Dressings.” Journal of Wound Care 7:246-50, 1998.
128. Langemo, D.K., Anderson, J., Hanson, D., et al. “Managing Fungating Wounds.” Advances in Skin & Wound Care 20(6):312-4, 2007.
129. Alexander, S. “Malignant Fungating Wounds: Epidemiology, Aetiology, Presentation and Assessment.” Journal of Wound Care 18:273-4, 276-8, 280, 2009.
130. Cooper, R., Molan, P. “The Use of Honey as an Antiseptic in Managing *Pseudomonas* Infection.” Journal of Wound Care 8(4):161-4, 1999.
131. Molan, P.C. “Re-introducing Honey in the Management of Wound and Ulcers—Theory and Practice.” Ostomy/Wound Management 48:28-40, 2002.
132. Cameron, J., Powell, S. “Contact Kept to a Minimum.” Nursing Times Wound Care Supplement 92(39):85-6, 1996.
133. Grocott, P., Robinson V. “Skin Problems in Palliative Care—Nursing Aspects.” In G. Hanks, N. Cherny, N. Christakis, et al., eds. The Oxford Textbook of Palliative Medicine, 4th ed. Oxford, UK: Oxford University Press, 2010:961-72.
134. Pudner, R. “The Management of Patients with Fungating or Malignant Wounds.” Journal of Community Nursing 12(9), 1998. Retrieved from http://www.jcn.co.uk.backiss.asp?YearNum=1998&MonthNum=09&ArticleID=82. Last Accessed 15 June 2014.
135. Baranoski, S. “Wound and Skin Care, Choosing a Wound Dressing, Part 2.” Nursing 38(2):14-5, 2008.
136. White, R., Cutting, K.G. “Modern Exudates Management: A Review of Wound Treatments.” World Wide Wounds, 2006. Retrieved from http://www.worldwidewounds.com/2006/September/White/Modern-Exudate-Management.html. Last accessed 20 May 2014.
137. Barton, P., Parslow, N., Savage, P. “Malignant Wound Management: A Patient-centered Approach.” In D.L. Krasner, et al. eds. Chronic Wound Care: A

Clinical Sourcebook for Healthcare Professionals, 4th ed. Wayne, PA: HMP Communications, 2008.
138. Bale, S., Tebbie, N., Price, P. “A Topical Metronidazole Gel Used to Treat Malodorous Wound.” British Journal of Nursing 13:S4-11, 2004.
139. Cutting, K. Wound and Infection Education Leaflet. Huntingdon, UK: Wound Care Society, 1998.
140. Merz, T., Klein, C., Uebach, B., et al. Fungating Wounds—Multidimensional Challenge in Palliative Care. Breast Care (Basel, Switzerland) 6(1):21-4, 2011.
141. Goldberg, M., Tomaselli, N.L. “Management of Pressure Ulcers and Fungating Wounds.” In A.M. Berger, R.K. Portenoy, D.E. Weissman, eds. Principles and Practice of Palliative Care and Supportive Oncology, 2nd ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2002:321-2.
142. Williams, C. “Clinisorb Activated Charcoal Dressing for Odour Control.” British Journal of Nursing 8(15):1016-9, 1999.
143. Adderly, U.J., Smith, R. “Topical Agents and Dressings for Fungating Wounds.” Cochrane Database of Systematic Reviews 2, 2007.
144. Dowsett, C. “Malignant Fungating Wounds: Assessment and Management.” British Journal of Community Nursing 8:394-400, 2002.
145. Haisfield-Wolfe, M.E., Rund, C. “Malignant Cutaneous Wounds: A Management Protocol.” Ostomy/Wound Management 43(1):56-60, 62, 64-6, 1997.
146. Bird, C. “Managing Malignant Fungating Wounds.” Professional Nurse 15(4):253-6, 2000.
147. Smith, S. Skin Care Following Radiation Therapy: The Clinician’s Notebook. Irving, TX: Carrington Laboratories, Inc. Newsletter; 1(3):1-3. Retrieved from http://www.woundcare.org/newsvol2n2./ar3.htm. Last accessed June 4, 2014.
148. Naylor, W. “Using a New Foam Dressing in the Care of Fungating Wounds.” British Journal of Nursing 10(Suppl 6):S24-30, 2001.
149. Mortimer, P.S. “Management of Skin Problems: Medical Aspects.” In D. Doyle, et al. Oxford Textbook of Palliative Medicine, 2nd ed. Oxford, UK: Oxford University Press, 1998.
150. Beare, P.G., Myers, J.L. Adult Health Nursing, 3rd ed. St. Louis, MO: CV Mosby, 1998.
151. Draper, C. “The Management of Malodour and Exudate in Fungating Wounds.” British Journal of Nursing 14(11):S4-12, 2005.
152. Mudge, E.J., Meaume, S., Woo, K., et al. “Patients’ Experience of Wound-Related Pain: An International Perspective.” European Wound Management Association Journal 8(2):19-28, 2008.
153. Ferris, F.D., Krasner, D., Sibbald, R.G. “12 Toolkits for Successful Wound Care. A Case-based Approach.” Journal of Wound Care 9:4-9, 2000.
154. Bryant, R. “Skin Pathology.” In N. Bryant, ed. Acute and Chronic Wounds: Nursing Management. New York: Mosby Year Book, 1992.
155. Hunter, S., Langemo, D.K., Thompson, P., et al. “Radiation Wounds.” Advances in Skin & Wound Care 20(8):438-40, 2007.

156. Black, J.M., Black, S.B. “Surgical Wounds, Tubes, Drains.” In S. Baranoski, E.A. Ayello, eds. Wound Care Essentials: Practice Principles. Philadelphia, PA: Lippincott Williams & Wilkins, 2008.
157. Subramania, I., Balasubramanian, D. “Management of Radiation Wounds.” Indian Journal of Plastic Surgery 45(2):325-31, 2012.
158. Mendelsohn, E., et al. “Wound Care After Radiation Therapy.” Advances in Skin & Wound Care 15(5): 216-24, 2002.
159. Ferris, F.D., et al. Palliative Wound Care: Managing Chronic Wounds Across Life’s Continuum: A Consensus Statement from the International Palliative Wound Care Initiative, 2004. Retrieved from http://www.palliativewoundcare.info. Last accessed 22 June 2014.
160. Morrison, R.S. “Palliative Care Outcomes Research: The Next Steps.” Journal of Palliative Medicine 8(1):13-5, 2005.
161. Chrisman, C.A. “Care of Chronic Wounds in Palliative Care and End-of-Life Patients.” International Wound Journal 7(4):214-35, 2010.
162. Langemo, D.K., Melland, H., Hanson, D., et al. “The Lived Experience of Having a Pressure Ulcer: A Qualitative Study.” Advances in Skin & Wound Care 13:225-35, 2000.

27

Wound Gallery

Sharon Baranoski, MSN, RN, CCNS-APN, CWCN, MAPWCA, FAAN

Elizabeth A. Ayello, PhD, MS, BSN, RN, CWON, ETN, MAPWCA, FAAN

1. What is the most likely diagnosis of this recently débrided ulcer on the plantar surface of the foot?

A. Pressure ulcer
B. Arterial ulcer
C. Diabetic ulcer

D. Venous ulcer

2. **Using the International Skin Tear Advisory Panel (ISTAP) classification system, what type of skin tear is seen here on the upper extremity?**

A. Type I
B. Type 2
C. Type 3
D. Type 4

3. **Given that this patient has gangrene of his forefoot, what is the most likely etiology?**

A. Arterial disease
B. Venous disease
C. Necrotizing fasciitis
D. Pyoderma gangrenosum

4. During a skin assessment, you discover the brownish-purplish dry area on the upper extremities; you suspect that this is:

A. melanoma.
B. pyoderma.
C. basal cell carcinoma.
D. senile purpura.

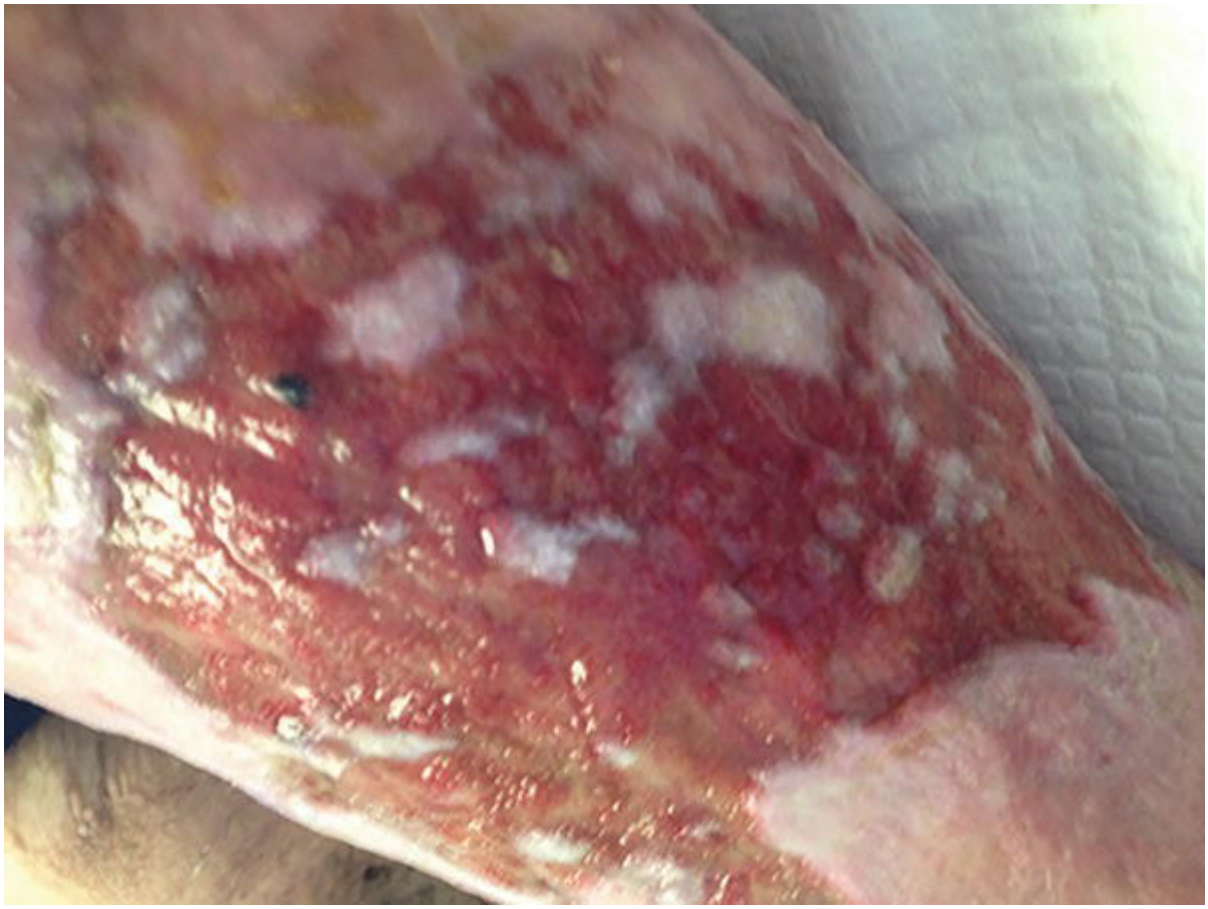
5. Using the National Pressure Injury Advisory Panel (NPIAP), this ulcer is classified as:

A. stage I.
B. stage III.
C. unstageable.
D. SDTI.

6. What do you think is the etiology of these multiple wounds on the fingers?

A. Frostbite
B. Eczema
C. Drug injection injury
D. Necrotizing fasciitis

7. What are these white areas of tissue called?



A. Biofilm
B. Granulation island buds
C. Slough
D. Eschar

8. What do you think is the underlying etiology of this wound?

A. Pressure
B. Renal failure
C. Sickle cell disease
D. Ischemia

9. Which of the following best describes this patient's legs?

A. Lymphedema with fibrosis
B. Lipedema
C. Melanoma
D. Scleroderma

10.What is the *primary* etiology of this skin injury?

A. Pressure ulcer
B. Skin tear
C. Gluteal cleft injury
D. Moisture-associated skin damage

11. This patient presents with ulcerative lesions and provides a history of working as an itinerant field laborer. What is the most likely diagnosis?

A. Buruli ulcer
B. Blastomycosis
C. Montezuma ulcer
D. Traumatic abrasions

12.Based upon the tissue appearance and clinical findings, the BEST diagnosis is:

A. gas gangrene.
B. infected postsurgical wound.
C. necrotizing cellulitis.
D. necrotizing fasciitis.

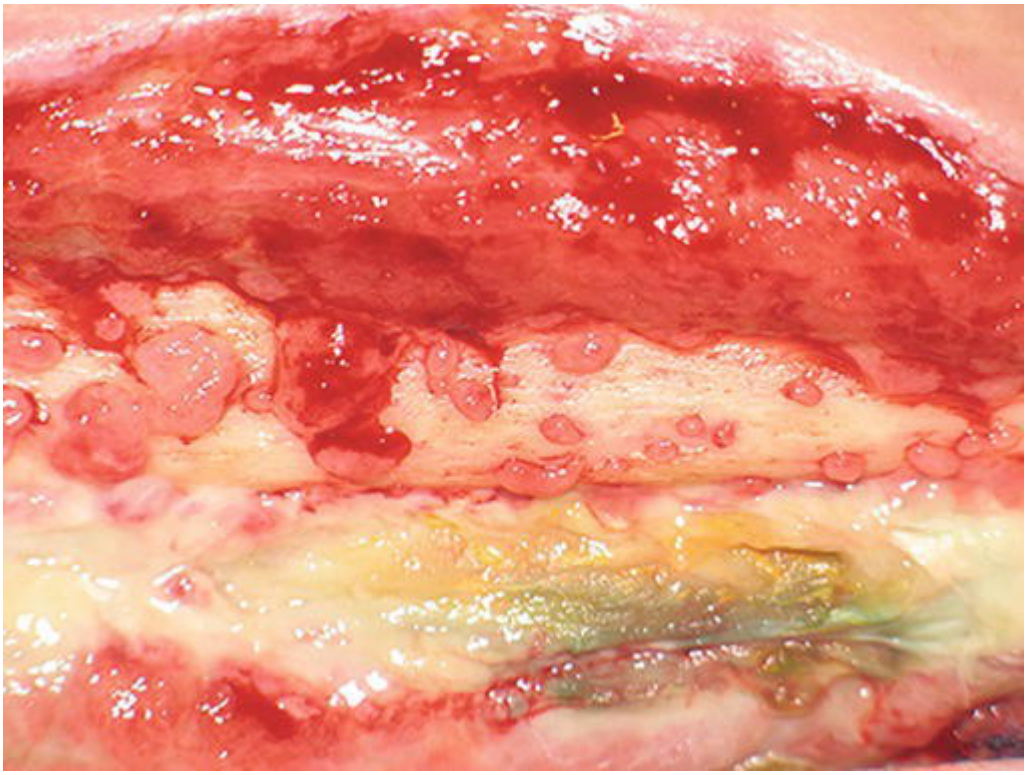
13.Which of the following is the most likely diagnosis?

A. Frostbite
B. Arterial “blue” toe
C. Subungual hematoma
D. Fungal infection

14.Which of the following is the best description of the translucent tissue indicated by the arrow?

A. Fibrin
B. Granulation
C. Neoepithelium
D. Proliferative

15.Granulation tissue that forms on exposed bone is unusual and suggests:



A. intact and viable periosteum.
B. recent revascularization.
C. use of hyperbaric oxygen.
D. application of negative pressure wound therapy.

16.Which of the following best describes these bilateral ulcers?

A. Sickle cell
B. Arterial
C. Venous
D. Lymphedema

17.This rapidly progressive and painful ulcer is highly suspicious for:

A. pyoderma gangrenosum.
B. basal cell carcinoma.
C. calciphylaxis.
D. vasculitis.

18.Which wound diagnosis is most accurate for this end-stage renal dialysis patient?

A. Calciphylaxis
B. Calcific uremic arteriolopathy
C. Uremic ulcers
D. Both A and B

19.Which noninvasive test is demonstrated in this photo?

A. Dermal thermography
B. Pulse oximetry
C. Transcutaneous oximetry
D. Skin perfusion pressure

20.This patient presents with a wound that is BEST classified as which of the following?

A. Arterial ulcer
B. Venous ulcer
C. Pressure ulcer
D. Atypical ulcer

21.How would you document the percentage of slough tissue in this pressure ulcer?

A. 10%
B. 25%
C. 30%
D. 90%

22.Would compression therapy be indicted for this patient?

A. Yes
B. No

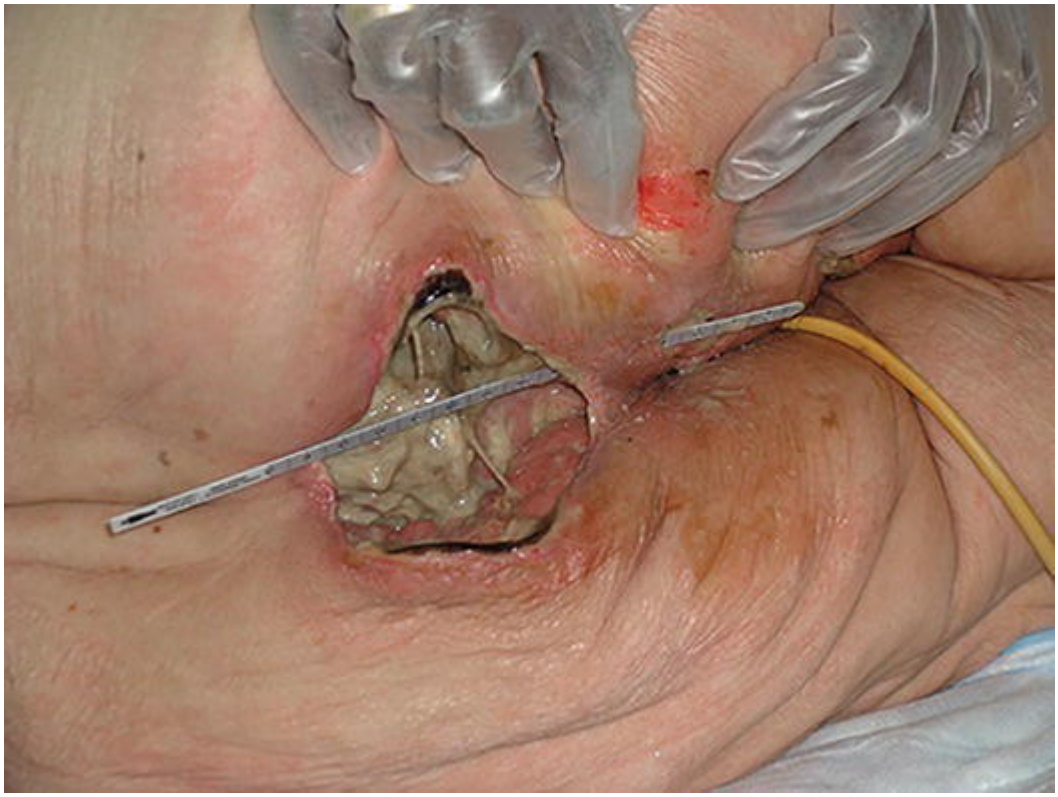
23.How would you document this wound type?

A. Suspected DTI
B. Hematoma
C. Necrotizing fasciitis
D. Unstageable pressure ulcer

24.What should be included in the plan of care for this patient?

A. Antibiotics
B. Maintenance débridement
C. Pressure off-loading
D. All of the above

25.This wound has a tunnel that should be documented. What position should be used to document the tunneling in this wound?



A. 1:00
B. 3:00
C. 5:00
D. 8:00

26.How would you describe the tissue at the wound edges?

A. Epithelialized wound edges
B. Rolled wound edges
C. Stalled wound edges
D. Necrotic wound edges

27.A priority for initiating treatment of this wound would be:

A. applying a dry dressing.
B. applying an emollient.
C. débriding of slough.
D. pending amputation.

28.What structure do you see in this wound?

A. Bone
B. Tendon
C. Muscle
D. Cartilage

29.Progressive degeneration of a weight-bearing joint, as depicted in this photo, is called:

A. Charcot foot.
B. hemosiderin deposit.
C. senile purpura.
D. pressure ulcer.

30.What is the correct documentation for this flap reconstruction called?

A. Pedicle flap
B. Bone flap
C. Split-thickness flap
D. Express flap

31.Extravasation caused by a chemotherapy agent resulted in this wound. What structure do you see that could impact the healing process?

A. Slough
B. Tendon exposure
C. Eschar
D. Cartilage

32.This wound is marked up to show areas of destruction surrounding the wound. This destruction is called:

A. slough.
B. tunneling.
C. hypergranulation.
D. undermining.

33.What diagnostic test does this photo represent?

A. Stemmer’s testing
B. Monofilament testing
C. Ankle–brachial index testing
D. Waveform testing

34.What type of tissue is primarily depicted in this wound bed?

A. Necrosis
B. Eschar
C. Slough
D. Granulation

35.What's wrong with this picture?

A. Nothing
B. Inappropriate sizing of compression stocking

36.How would you describe the discoloration around this wound?

A. Hemosiderin deposits
B. Contact dermatitis
C. Xerosis
D. Gangrene

37.The type of dressing seen in this photo is called:

A. collagen dressing.
B. hydrocolloid dressing.
C. hydrogel dressing.
D. foam dressing.

38.A rare but severe surgical complication where the incision opens and the internal muscle/organs and/or hardware can be seen in the incision is called:

A. deep tissue injury.
B. dehiscence.
C. evisceration.
D. necrotic tissue.

39.What type of dressing would be appropriate for this wound?

A. Hydrogel
B. Foam
C. Dry gauze
D. Betadine mesh

40.Patient presents with a(n) _____ to his lower right foot.

A. skin tear
B. blister
C. DTI
D. evacuated hematoma

41.The etiology of this left lateral foot wound is probably:

A. arterial.
B. venous.
C. pressure injury.
D. diabetic.

42.This skin tear is classified as:

A. type 1.
B. type 2.
C. type 3.
D. type 4.

43.This Achilles wound developed due to:

A. medical device.
B. diabetes.
C. sickle cell.
D. squamous cell.

44.Treatment options for this foot should include:

A. off-loading.
B. callus débridement.
C. anhydrous management.
D. off-loading and callus débridement.

45.What type of ulcer is this?

A. Arterial
B. Venous
C. Sickle cell
D. Squamous cell cancer

46.How should you document these feet?

A. Normal finding
B. Fungus infection
C. Yeast infection
D. Charcot foot

47.What is the gold standard of treatment for this venous ulcer?

A. Ace wraps
B. Roller gauze
C. Compression wraps
D. Intermittent pneumatic therapy

48.This genetic blistering disease is most likely:

A. hidradenitis suppurativa.
B. bullous pemphigoid.
C. acanthosis nigricans.
D. epidermolysis bullosa.

49.How should these painful swollen digits be documented?

A. Dactylitis
B. Frostbite
C. Arthritis
D. Scleroderma

50.How should this pressure injury be classified?

A. Stage 1
B. Stage 2
C. Unstageable
D. Deep pressure injury

Thank you to our colleagues for this chapter and previous editions of this chapter for their contributions of photos. Photo acknowledgments for this chapter:

Photo 1: Javier La Fontaine, DPM, MS

Photo 2, 39: Mary Gloeckner, RN

Photos 3, 34, 45, and 47: Mary Y. Sieggreen, MSN, APRN, CS, CVN, APN

Photo 4: Beth Beck, RN

Photos 5to 10, 43: Jeffrey M. Levine, MD, AGSF, CMD, CWSP

Photos 11 to 12, 14 to 15, 17 to 20, 40, and 44: Jeffrey A. Niezgoda, MD, FACHM, MAPWCA, CHWS

Photos 13 and 46: K. Zinszer, DPM

Photos 16 and 41: R. Gary Sibbald, MD, M.Ed, M.Sci (Hon), MAPWCA

Photos 21 to 28 and 32: KDS Professional Consulting

Photos 29, 33, and 35: Elizabeth A. Ayello, PhD, RN, ACNS-BC, CWON, MAPWCA, FAAN

Photos 30 to 31, 36 to 37, 42, and 50: Sharon Baranoski, MSN, RN, CCNS-APN, CWCN, MAPWCA, FAAN

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APPENDIX

Definitions of the EPUAP/NPIAP/PPPIA 2019 Clinical Guideline Strengths of Evidence and Strengths of Recommendations

The groups that worked on the 2019 EPUAP/NPIAP/PPPIA Clinical Guideline assigned a level of evidence for each study they read based on criteria listed in the first table below. Groups then used a consensus voting process to assign a strength for each of the recommendations. The second table lists the explanation for the strengths of recommendation. The strength of the recommendation is intended to be used by health professionals to help them prioritize interventions.

Strengths of Evidence Used in the EPUAP/NPIAP/PPPIA 2019 Clinical Guideline

Strengths of Evidence	
A	• More than one high-quality level 1 study providing direct evidence • Consistent body of evidence
B1	• Level 1 studies of moderate or low quality providing direct evidence • Level 2 studies of high or moderate quality providing direct evidence • Most studies have consistent outcomes and inconsistencies can be explained
B2	• Level 2 studies of low quality providing direct evidence • Level 3 or 4 studies (regardless of quality) providing direct evidence • Most studies have consistent outcomes and inconsistencies can be explained
C	• Level 5 studies (indirect evidence), e.g., studies in normal human subjects, humans with other types of chronic wounds, animal models • A body of evidence with inconsistencies that cannot be explained, reflecting genuine uncertainty surrounding the topic
GPS	**Good Practice Statement** • Statements that are not supported by a body of evidence as listed above but considered by the GGG to be significant for clinical practice

Strengths of Recommendation Used in the EPUAP/NPIAP/PPPIA 2019 Clinical Guideline

Strengths of Recommendation	
↑↑	Strong positive recommendation: Definitely do it
↑	Weak positive recommendation: Probably do it
↔	No specific recommendation
↓	Weak negative recommendation: Probably don't do it
↓↓	Strong negative recommendation: Definitely don't do it

European Pressure Ulcer Advisory Panel, National Pressure Injury Advisory Panel, and Pan Pacific Pressure Injury Alliance. In E. Haesler, ed. *Prevention and Treatment of Pressure Ulcers/Injuries: Clinical Practice Guideline. The International Guideline 2019*. EPUAP/NPIAP/PPPIA, 2019.

For a full explanation of the methodology, please consider accessing the digital or print copy of the guideline. Information on how to access the guideline is available from the following Web sites:

NPIAP Web site www.npiap.com

EPUAP Web site www.epuap.org

PPPIA Web site www.pppia.org

International Pressure Injury Guideline Web site
www.internationalguideline.com

APPENDIX

Answers

Chapter 1

1. **C.** Those afflicted with wounds are often viewed as appalling and repulsive.
2. **D.** Assessment of pain is commonly lacking in wound assessment; size, odor, and drainage are usually assessed.
3. **A.** The patient's perceptions of well- being should direct quality-of-life treatment decisions.
4. **D.** All of the above.

Chapter 2

1. **C.** Answer A is incorrect because it refers to the Medicaid program, which is a collaboration between the federal and state governments to deliver care. Answer B is incorrect because Medicare Part A is for inpatient hospital costs. Answer D is incorrect because Medicare Part B requires the beneficiary to pay a 20% copayment.

2. **B.** OASIS-C is only used by home health agencies to assess patients and determine reimbursement.

3. **A.** Answer B is incorrect because the beneficiary must spend 3 consecutive days in the hospital. Answer C is incorrect because skilled nursing services must be needed on a daily basis. Answer D is incorrect because the beneficiary must be admitted within 30 days of hospitalization.

4. **C.** There are currently no quality initiatives by CMS on turning patients. Answers A, B, and D are all true with regard to CMS. A is true because data on the performance of hospitals, nursing homes, and home health agencies are readily available on the CMS Web site. B is true because there is tremendous interest at CMS to reward providers of high-quality care, known as pay for performance. D is true because QIOs are emphasizing working with providers to improve care rather than just detecting episodes of poor care.

Chapter 3

1. **C.** The jury determines who, what, when, why, where, and how—in other words, what happened. The judge interprets the law by instructing the jury about the law to be applied to the facts as it has determined them. The other options are incorrect.

2. **D.** The plaintiff must prove all four elements in order to prevail at trial.

3. **A.** Opinions related to patient care aren't proper entries in a medical record. The medical record is a communication tool among practitioners and is best used for the transmittal of factual information. The other options are incorrect.

4. **A.** Answer A is the correct choice as it is the one that is false. Standards must be reviewed and amended to reflect the latest research and practice experience in a treatment area.

5. **C.** Suspected DTI damage may occur days before it is visibly evident on the patient. The other options are incorrect.

6. **D.** An individualized turning schedule based on patient assessment q4h turning is the best way to document the frequency of turning in the medical record. The other options are incorrect because they are rigid and inflexible and do not allow for individualized patient care. Too many variables can impact repositioning to mandate an absolute time frame for all patients.

7. **A.** Normative policies and procedures describe care that can be realistically and consistently provided. The other options are incorrect. Answer B is the definition of positive policies and procedures; answer C, policies are not based on staffing ratios; and answer D has nothing to do with normative policies.

Chapter 4

1. **D.** The cells of the stratum corneum can shed and look like flakes during routine cleaning activities such as bathing.
2. **D.** Upon stimulus from the hypothalamus, skin blood vessels will either vasoconstrict (heat needs to be conserved to elevate temperature) or vasodilate (heat needs to be eliminated to lower temperature) depending upon specific needs. Skin can synthesize vitamin D, not vitamin K. Carbon dioxide is eliminated via the lungs. Glucose levels are regulated by the islets of Langerhans in the pancreas, not the Langerhans cells in the skin.
3. **D.** The entire biology of keratinocytes is dedicated to barrier formation and its maintenance.
4. **D.** ABI is a test used for peripheral vascular disease; it does not tell you about skin assessment. Answers A, B, and C should all be part of a skin assessment.
5. **D.** Emollient moisturizers are a cornerstone in the treatment of xerosis. Answer A is incorrect because daily cleaning of the skin by either showering or bathing is not recommended as it further dries the skin. Answer B is incorrect because a low-pH soap needs to be used as deodorant soaps have a high pH that makes the skin alkaline. Answer C is incorrect because rubbing can irritate dry skin.
6. **C.** Pruritus is the medical term for itchy skin.
7. **D**. Bulla. Answer A is incorrect because a papule is an elevated, palpable, firm, circumscribed lesion up to 1 cm. Answer B is incorrect. Although a vesicle is a fluid-filled blister, it is only up to 1 cm. Answer C is incorrect as although it is elevated and similar to a vesicle, it is filled with pus.

Chapter 5

1. **B.** Redness, pain, swelling, and heat are the four cardinal signs of the normal inflammatory response. However, the presence of bleeding is more indicative of local infection in a wound as granulation tissue should not bleed easily to light touch.

2. **E.** Wound biofilms can be effectively treated by a combination of debridement and/or cleansing to remove the biofilms, followed by application of dressings that block new bacteria from reaching the wound and killing bacteria left in the wound bed. This concept for treatment of chronic wounds that likely have biofilm bacteria has been termed “biofilm-based wound care.”

3. **D.** Symptoms frequently accompany individuals with chronic wounds. Many patients experience four or more concurrent symptoms, which frequently include pain, fatigue, anxiety, depression, and sleep disturbances.

Chapter 6

1. **D.** Wound assessment involves observation, data collection, and an ongoing evaluation process. Surgical debridement is an intervention for the management of wound care.

2. **B.** A superficial wound involves only the epidermis and the dermis is still intact. A full-thickness wound extends through the dermis. A subdermal wound extends into underlying structures below the skin such as bone, muscle, or tendon.

3. **D.** A sinus tract/tunnel is a channel that involves an area larger than the visible surface of the wound. Maceration is the softening of the surrounding skin usually from exposure to or excess wound drainage. A fistula is an opening between two organs or between an organ and the skin.

4. **C.** Holding the camera at a 90-degree angle rather than perpendicular to the wound plane can cause distortion of the wound size in the photo. Answers A, B, and D are correct techniques that should be part of a wound photo protocol.

5. **A.** Electronic health records and electronic medical records are different. The electronic medical record is a legal record within a special institution, such as a hospital, while electronic health record uses the electronic medical record as a source of information to share patient data across care settings. Answers B, C, and D are true.

Chapter 7

1. **D.** Antiseptics are an integral part of chronic wound treatment. They are effective in killing a range of planktonic organisms but are not site specific so sensitivity to the drug is not an issue. Antiseptics reduce the number of planktonic microorganisms on the wound surface, resulting in lower bioburden and enhanced healing. The most ideal antiseptics have low toxicity.

2. **C.** Semiquantitative cultures give accurate information and are most frequently used; infection is indicated by 4+ rating. Quantitative cultures are available primarily in research institutions. The specific organisms in question can be identified with either approach.

3. **D.** Biofilm cannot be visualized. While slough is visible, slough is not diagnostic of biofilm as it only provides a surface for the growth of organisms. Biofilm is not uniformly distributed, making culture of it challenging. Biofilm is difficult to treat with antibiotics.

4. **C.** Data support weekly sharp debridement because of its ability to remove the biofilm, the media it lives on, and stimulate an acute response to healing. Often sharp debridement is not used because of lack of trained personnel to perform it. However, it is important to recognize that the various noninvasive approaches are not as effective as sharp debridement.

Chapter 8

1. **B.** Debridement is the removal of debris so that cell movement can be enhanced. Answer A is incorrect because most wounds need debridement to heal; answer C is incorrect because removing necrotic tissue decreases not increases the wound's biological burden; and answer D is incorrect because healable wounds need moist healing.

2. **A.** Exudate is a sign of infection, which signals the need for debridement. Other signs are erythema and fluid wave. The other options are incorrect.

3. **D.** Pulsed lavage is a method of mechanical debridement. Answer A is incorrect because collagenases are enzymes; answer B is incorrect because maggots secrete natural collagenase; and answer C is incorrect because film dressings are a method of autolytic debridement.

4. **A.** Because the resident is on Coumadin and bleeding can occur, surgical debridement would be least indicated. Also, the appropriate personnel and equipment may not be available in the patient's long-term care facility. The other options are correct and should be considered within the context of other patient characteristics.

5. **A.** Time is of the essence, and surgical debridement is the quickest method that can be used with infected wounds. Because the client is hospitalized, the appropriate personnel and equipment to perform this method of debridement are available. The other options are incorrect.

Chapter 9

1. **B.** Answers A, C, and D are all old concepts of wound management.
2. **C.** Gauze is a form of dry dressing therapy. Answers A, B, and D are moist wound therapy dressings.
3. **B.** Nurse preference shouldn't be a parameter of dressing selection. Answers A, C, and D are appropriate dressing parameters.
4. **A.** Transparent film doesn't absorb fluid. Answers B, C, and D are all advantages of transparent film use.
5. **A.** Answer A is true. This is a fact question supported by the data in the chapter.
6. **D.** Apligraf is a bilayered skin substitute. Answers A, B, and C are all moist wound therapy choices.
7. **D.** Hydrogel amorphous gel has minimal ability to absorb drainage, so it is not indicated for use in heavily draining wounds. Answers A, B, and C are all specifically used for heavily draining wounds.
8. **A.** Tissue-engineered skin substitutes must be applied to a wound bed that is free of necrotic tissue and infection. Answers B and C are incorrect as rejection of these products is not an issue. Although the patient's weight may be part of the total care plan, it is not essential to evaluate for use of this product.
9. **B.** Tissue oxygenation is increased with the use of electrical stimulation. Answers A, C, and D are all correct.
10. **C.** Hyperbaric oxygen therapy causes vasoconstriction not vasodilation in both arterial and venous vessels. Answers A, B, and D are all ways in which hyperbaric oxygen therapy enhances wound healing.

Chapter 10

1. **C.** 85 to 102 g. 149÷2.2 = 67.7 kg × 1.2–1.5 = 79.7. This is within the daily protein requirement of 68-86 g.
2. **B.** Request RDN referral to assess her nutritional status and ask the physician to consider ordering a regular diet.
3. **C.** High-protein, high-calorie supplement with arginine and micronutrients between meals.
4. **B.** Offer 30 to 35 kcal/kg body weight daily for adults with a pressure injury.
5. **A.** Arginine and glutamine.

Chapter 11

1. **D.** Lateral rotation moves side to side to aid pulmonary function, but unless a second feature is present such as low air loss, it's not appropriate for pressure ulcer prevention.
2. **D.** All are important considerations. A patient at home may have different needs than one in an acute care setting.
3. **C.** Support surfaces redistribute pressure.
4. **D.** This is the National Pressure Injury Advisory Panel definition of pressure redistribution. Immersion is the depth of penetration into a support surface. Envelopment is the ability of a support surface to conform to the body irregularities. Pressure gradient is a change in pressure over a distance.

Chapter 12

1. **D.** McCaffery's classic definition of pain is that it's whatever the experiencing person says it is. Answer A is incorrect as research has shown that sudden, severe pain may elevate vital signs, but this only occurs for a short time. Answer B is incorrect as research has shown that patients can sleep even though they have moderate or severe pain. Answer C is incorrect because even though pain may be a consequence of wound care, this isn't a definition of pain.

2. **A.** The Numeric Pain Intensity Scale is a 10-cm line with the words "no pain" at one end and "worst possible pain" at the other end. Answer B refers to the Faces Pain Rating Scale. Answer C doesn't describe a pain scale. Answer D refers to the WHO analgesic ladder.

3. **B.** A nonopioid is the drug recommended initially for mild pain. An adjuvant can be added if there is neuropathic and/or nociceptive pain. Answer A is wrong as drugs are part of the WHO analgesic ladder. Answers C and D are incorrect as they are part of step 2 in the ladder.

Chapter 13

1. **D.** A 72-year-old female is the oldest, least mobile, and receiving steroids, which are known to further cause thinning of the skin, so her skin is at highest risk.

2. **B.** Answer A is incorrect because there is no tissue loss in type 1. Type 3 is incorrect because in category III, there is complete loss of the tissue flap. Answer D is incorrect because type 4 is not part of the classification system.

3. **A.** Nonemollient soaps should be used instead of detergent, which dries the skin. The literature suggests that routine every-other-day bathing for elderly people is adequate (unless the skin is soiled) and can reduce skin tear injury. Answers B, C, and D are all interventions to consider as part of a skin tear protocol.

4. **D.** While all four choices are types of moisture-associated skin damage (MASD), the etiology of all four is different. It is important to differentiate between them. Intertriginous dermatitis (ITD) is the type of MASD caused by trapped perspiration and frictional forces from opposing skin surfaces. While various microorganisms can proliferate, of those which are fungal, *Candida* can be found in ITD. Answer A is incorrect because peristomal skin damage occurs around an ostomy, and there was no information that this patient had a stoma on their abdomen. Answer B is incorrect, because there was no information that the patient had an abdominal wound. Answer C is incorrect because there is no information that the patient was incontinent of urine or stool.

5. **B.** Chronic plaque psoriasis is the most common variant form of psoriasis. Answers A, C, and D are forms of psoriasis but NOT the most common variant.

6. **B.** This is the definition of skin failure. The other answers are wrong. Xerosis is dry skin, Pruritus is itchy skin and Skin tear is a separation of skin layers from trauma.

7. **D.** 80 g/dL—all others are too low.

8. **B.** Dystrophic. Squamous cell carcinoma is not seen in A and C (simplex and kindelar); Koebner is a type of psoriasis.

Chapter 14

1. **B.** Unrelieved pressure is the cause of tissue death in pressure ulcers. Incontinence and diabetes mellitus are patient characteristics that may put a patient at risk for pressure ulcers but in and of themselves don't cause pressure ulcers. Heat causes burns, not pressure ulcers.

2. **C.** Friction has occurred. Electrical stimulation is an adjunct therapy used to heal pressure ulcers. Shear is a type of mechanical trauma caused by tissue layers sliding against each other. Maceration is not a mechanical force caused by dragging the skin across a surface.

3. **C.** In this ulcer, the tissue destroyed is into the subcutaneous tissue. The newly revised NPIAP classification system now includes that category/stage 3 pressure ulcers are full-thickness wounds that may have some slough and undermining/tunneling, but bone or muscle is not visible or palpable. The ulcer isn't a stage 1 because the epidermis is no longer intact. It isn't a stage 2 because the tissue destroyed is deeper than superficial level (partial thickness) and is well into the subcutaneous tissue and it has slough, there is no slough in a stage 2 pressure ulcer. It isn't a stage 4 because in this ulcer, muscle or bone is not palpable or visible.

4. **D.** The research-based cut score for onset of pressure ulcer risk for older patients and blacks is 18. With the Braden Scale, scores at or lower than the cutoff score indicate risk for pressure ulcer development. Answers A, B, and C are all wrong answers because they're higher than the cutoff score of 18. With the Braden Scale, low numerical scores indicate a risk for pressure ulcers.

5. **C.** It is recommended that pressure lifts be done every hour for chair-bound patients. Answer A is incorrect. The exact frequency of turning a patient is not yet known but needs to be individualized. Turn the patient at least every 2 to 4 hours as his or her condition warrants. Answer B is incorrect. Don't

use hot water, but rather warm water, and avoid soaps that dry the skin. Answers D is incorrect as there's no need to limit the patient's fluids.

6. **A.** Depth is not on the PUSH tool to measure pressure ulcer healing. Exudate, tissue type, and length × width are the three variables measured.

7. **B.** No. Mucosal pressure injuries cannot be staged using the NPIAP staging system because mucosa does not keratinize.

Chapter 15

1. **B.** Venous stasis was thought to cause venous ulcers because of pooled blood in the veins. However, current literature reports that venous hypertension is responsible for increased pressure along the vein wall and in the subcutaneous tissue.
2. **A.** Perfusion of the lower extremity is indirectly measured by the ABI.
3. **C.** Compression is the most important component—the edema must be managed in order for venous and lymphatic ulcers to heal.
4. **C.** Nicotine shouldn't be used in any form. It constricts vessels and contributes to atherosclerosis and venous disease.

Chapter 16

1. **B.** CDT: Complete Decongestive Therapy—this two-phase form of treatment consists of:

1. Decongestive/intensive phase of reducing the limb with manual lymphatic drainage, short stretch bandages, meticulous skin and nail care, and home exercise program.
2. Maintenance phase of transitioning the limb to a proper fitting day and night maintenance compression garment, continuing with home exercise program and meticulous skin and nail care.

2. **B.** False: Graded compression should not be worn in supine position for extended periods as it addresses working pressure.

3. **D.** There are four stages of lymphedema, they include:

Stage 0 Lymphedema/subclinical/latency lymphedema: the earliest stage of lymphatic impairment. Lymph transport is afflicted; however, there are no signs of edema. Subjective complaints may include “tight, full, heavy, achy.”

Stage 1 Lymphedema/early-onset/reversible lymphedema: this stage includes presence of edema and possible pitting, but changes in tissue integrity are minimal. Swelling often responds to elevation in this stage.

Stage 2 Lymphedema/spontaneously irreversible lymphedema may be subdivided further into “early and late stage 2.” In early stage 2, pitting edema may still be present but tissue changes are occurring, and the limb is no longer responding to elevation. Late stage 2 demonstrates nonpitting edema with fibrosis.

Stage 3 Lymphedema/lymphostatic elephantiasis: Significant skin changes occur in this stage, and infections can be frequent. Presentation of the affected area includes deep skin folds, hypertrophy, papillomas, fibrotic tissue throughout, and disfigurement.

Chapter 17

1. **D.** Varicose veins of the lower legs are an early sign of venous insufficiency, and the presence of venous disease is not a known risk factor for the development of arterial disease. Smoking, hypercholesterolemia, diabetes mellitus, and hypertension are risk factors for arterial disease.

2. **C.** In patients without the ability to heal, antimicrobials that will work with moisture reduction, such as povidone–iodine or chlorhexidine, are a necessary treatment. Other treatments include conservative debridement of the slough, moisture reduction, and antibacterials that work in a dry environment. For silver to be effective in a wound bed, it requires moisture to be converted to the ionized form, and this is contraindicated in ulcers without the ability to heal.

3. **B.** An arterial ulcer by definition is always associated with arterial insufficiency or reduced blood flow (100%). Lipodermatosclerosis and edema are associated with venous ulcers. There is a less common but significant association of arterial ulcers with diabetes (30%) and hypertension in 29% to 39% of patients.

4. **A.** Perfusion of the lower extremity is indirectly measured by the ABPI.

5. **C.** Arterial ulcers are associated with arterial insufficiency, and a bypass graft is meant to restore the arterial circulation to the ischemic tissues. The other options are incorrect.

Chapter 18

1. **B.** One-third of people with diabetes are unaware of their condition.
2. **A.** Diabetes mellitus is the single leading cause of lower extremity amputation. Answers B, C, and D are not the single leading cause of amputation in diabetic patients. While these conditions can contribute to complications in the diabetic, they aren't listed as the leading cause for amputation.
3. **B.** Moisturizers shouldn't be applied between the toes—fungal infections can occur. All others are ADA recommendations for good skin care.
4. **A.** Off-loading must be tailored to the individual.
5. **D.** Studies by researchers have found that making walking boots irremovable (nonremovable) that patients had same healing as with the gold standard of off-loading, the total contact cast. The other options are all removable shoes, and research has not demonstrated healing rates comparable to TCC.

Chapter 19

1. **C.** Persons with heterozygous sickle cell disease receive only one gene for abnormal hemoglobin from one of their parents and are at less risk of ulceration than persons who have abnormal hemoglobin genes from both parents (homozygous). Answers A, B, and D are true statements.

2. **A.** Answer is true. This is a fact question supported by the data in the chapter.

3. **A.** Edema is a key indicator that a person with sickle cell disease may get an ulcer. Answer B is incorrect as hemoglobin A_{1C} is not related to sickle cell disease but to diabetes mellitus. Answer C is incorrect as patients with sickle cell disease often have fever of unknown origin. D. Pain is frequently a major problem for patients with sickle cell ulcers.

4. **D.** A bone scan is virtually useless in diagnosing osteomyelitis in the sickle cell ulcer patient while an MRI is preferred. Answers A, B, and C are all correct and should be part of the assessment of a patient with a sickle cell ulcer.

5. **E.** All of the above are all important parts of the holistic plan of care of patients with a sickle cell ulcer.

Chapter 20

1. **B.** If a wound has remained opened for a period of time and then heals through delayed surgical closure (e.g., flap, graft, primary closure) or by using an advanced modality, it is considered healing through tertiary intention.

2. **C.** Any dressing that is applied over the suture line should have the ability to protect the wound. The suture line should be protected for at least 48 hours until the natural skin barrier is typically formed.

3. **D.** There are three types of SSIs. The first type is superficial incisional SSI involving the skin only or the subcutaneous tissue of the incision. The second type is deep incisional SSIs involving the fascia and/or muscular layers. The last type is organ/space that involves any part of the body that has been opened or manipulated during the procedure except the skin incision, fascia, or muscle layers.

4. **B.** Secondary healing is the simplest method for any wound, but in many deep wounds, secondary healing may be lengthy and result in excessive scarring. Venous stasis ulcers and sternal dehiscence can sometimes heal secondarily, and skin grafting would be more complex.

5. **C.** Only the radiation of the chest wall is a wound with muscle involvement.

6. **D.** Arterial insufficiency in the flap is a major hazard, and problems can occur from too much tension on the artery, hematoma in the underlying tissue, extravasated blood, increased blood viscosity, spasm of the artery, or injury or disease of the arterial vessels. Monitor for the following signs of vascular compromise:Pallor and coolnessLack of pulsesPain with movementSlow or absent capillary refillInability to move extremity

7. **B.** The patient must be compliant with off-loading strategies after a surgical flap for wound closure to prevent breakdown of the surgical repair. This involves not only immediate

postoperative pressure redistribution but continued interventions of pressure relief and reduction after complete healing has occurred. Proper chair cushions and weight shifts are essential.

Chapter 21

1. **C.** A definitive indicator of a cutaneous fistula is the passing of GI secretions or urine into an open wound bed or through an unintentional opening onto the skin. Fever, erythema, wound dehiscence, pain, and exudate are indicators of possible infection and should be observed for further complications; however, the definitive indicator is GI contents or urine passing to the wound bed.

2. **C.** If the tube is stabilized, the skin is less likely to break down; if adequate hydration is maintained, there will be fewer problems with the tube; nutritional support is the primary goal.

3. **D.** Crohn's disease is an inflammatory bowel disease that affects all layers of the bowel wall, predisposing the patient to fistula formation. Answers A, B, and C are all within normal range.

4. **B.** Using a bedside gravity drainage system will prevent overfilling the pouch and reduce leakage. Answers A and C expose the skin to corrosive fistula drainage. Answer D runs the risk of dehydrating the patient and reducing caloric intake.

Chapter 22

1. B. It's critical to recognize when a wound is caused by an etiology other than prolonged pressure, neuropathy, or abnormal vascular supply so that appropriate measures may be undertaken to make a correct diagnosis and provide appropriate therapy. Although an infectious agent that's contagious may be a cause of an atypical ulcer, this isn't common. Although oftentimes specific therapies for atypical wounds exist, these are usually coupled with principles of good wound care, such as compression, off-loading, moist wound healing, and others. Billing for medical therapies is based on Evaluation and Management Codes as opposed to CPT codes.

2. **D.** As a variety of etiologies cause atypical wounds, a variety of techniques are used to confirm these etiologies. Histology is critical for diagnosing inflammatory, malignant, and infectious causes. Biopsies for tissue culture aid in diagnosing infectious causes, and biopsies for immunofluorescence will aid in diagnosing some inflammatory and autoimmune diseases.

3. **C.** In susceptible people, even minimal trauma to the skin can result in the production of pyoderma gangrenosum lesions, such as pustules and ulcers. This phenomenon is called pathergy. Therefore, debridement of an ulcer secondary to pyoderma gangrenosum may lead to severe worsening of the ulcer.

4. **A.** Sporothrix schenckii causes sporotrichosis. Mycobacterium ulcerans and M. marinum are species of mycobacteria. Fonsecaea pedrosoi is a pigment fungi related to chromoblastomycosis.

5. **C.** Cryofibrinogenemia is a painful cutaneous ulceration classified as a vasculopathy. Mycobacterium is a bacterium. Pyoderma gangrenosum is an inflammatory ulcer, and metabolic disease is an uncommon cause of chronic wounds.

6. **A.** Calciphylaxis is the correct answer. Vasculopathy, radiation dermatitis, and chemical burns are all other types of atypical wounds.

7. **B.** Factitial dermatitis is a self-imposed injury usually found in easily accessible areas such as the extremities, abdomen, and anterior chest. The other options are incorrect.

8. **B.** Vascular inflammation. Words ending in “it is” such as vasculitis means inflammation.

9. **A.** Thrombus is the process of a blood clot forming in a blood vessel. Extravasation is caused by Solutions of calcium, potassium, bicarbonate, hypertonic dextrose, cardiac drugs, chemotherapeutic drugs, cytotoxic drugs, and antibiotics can lead to extravasation injury. Venous insufficiency.

Chapter 23

1. **C.** Although more males have SCI, gender isn't a major risk factor for pressure ulcer development. Pre-existing conditions, severity of the SCI, and nutrition are all major risk factors for pressure ulcer development.

2. **B.** Urinary tract infection is the most commonly occurring complication of SCI. Pressure ulcer development is second. Fractures and osteoporosis are also other complications that may occur.

3. **D.** A is incorrect as the exact number of hours for a turning and repositioning schedule must be individualized for the person based on the type of support redistribution surface being used and the patient's conditions and comorbidities. B and C are correct and should be part of a prevention plan.

Chapter 24

1. **C.** Intertriginous dermatitis.
2. **B.** False. Acanthosis nigricans is hyperpigmented (often brown) irregular plaques in skin folds. This condition is associated with obesity as well as other systemic diseases, such as insulin resistance, diabetes, hormonal changes such as hypothyroidism, cancer, and more. In many situations, treating the underlying problem can help fade the discoloration. Examples may include weight loss, medication, or surgery to address the underlying cause. If the skin surface becomes aesthetically problematic or uncomfortable for the patient, consider prescription creams to lighten the area, antibacterial soaps to reduce odor, or laser therapy to reduce the thickness of the skin surface.
3. **D.** Supporting the pannus and other large skin folds.

Chapter 25

1. **C.** Maturity of the integumentary system is complete at approximately 33 weeks. At 24 weeks gestation the skin has minimal stratum corneum. Subcutaneous fat deposit begins to form (weeks 26 to 29).
2. **C.** Occiput is the most popular area for skin breakdown or pressure injury to occur. All of the rest are sites that need to be closely monitored.
3. **D.** The Braden Q scale and/or Glamorgan scale is the correct answer. The rest are adult scales.

Chapter 26

1. **A.** Palliative care is focused on holistically supporting an individual for comfort rather than care or healing of a wound while improving quality of both living and dying. Palliative care affirms life and views death as part of a normal process and is implemented to neither delay nor speed death. Palliative care provides relief from pain and other symptoms yet is not focused on the complete elimination of these symptoms. Answers B, C, and D do not define palliative care.

2. **D.** A hydrogel dressing that is placed on a wound every 3 days and as needed is recommended in a palliative care patient with a wound, as the hydrogel provides for a moist wound environment, which is soothing and comforting to the patient. It assists in protecting the periwound skin from maceration and is nonadherent, which is desirable. A silverimpregnated or calcium alginate dressing is not necessary unless there is heavy exudate or signs of infection or moderate to severe inflammation. Wet-to-dry dressings that are changed every 4 hours could likely contribute to periwound maceration and cause pain.

3. **B.** Warm rather than cold saline is recommended when irrigating wounds in palliative care patients. Answers A, C, and D are appropriate management strategies.

4. **C.** The skin is moist and desquamated, so it needs protection from injury caused by the edema and wetness. Answer A is incorrect because the skin is compromised and needs care. Answer B is incorrect as the skin is too moist already. Answer D is incorrect because debridement is not indicated. In addition, debridement agents do not need to be applied as frequently as three times per day.

Chapter 27

1. **C.** Diabetic ulcer.
2. **B.** Type 2.
3. **A.** Arterial disease.
4. **D.** Senile purpura.
5. **C.** Unstageable.
6. **C.** Drug injection injury.
7. **B.** Granulation island buds.
8. **D.** Ischemia.
9. **A.** Lymphedema with fibrosis.
10. **D.** Moisture-associated skin damage.
11. **B.** Blastomycosis.
12. **D.** Necrotizing fasciitis.
13. **C.** Subungual hematoma.
14. **C.** Neoepithelium.
15. **A.** Intact and viable periosteum.
16. **C.** Venous.
17. **A.** Pyoderma gangrenosum.
18. **D.** Both A and B.
19. **C.** Transcutaneous oximetry.
20. **B.** Venous ulcer.
21. **D.** 90%.
22. **B.** No.
23. **B.** Hematoma.
24. **D.** All of the above.
25. **C.** 5:00.

26. **A.** Epithelialized wound edges.
27. **C.** Debriding of slough.
28. **B.** Tendon.
29. **A.** Charcot foot.
30. **A.** Pedicle flap.
31. **B.** Tendon exposure.
32. **D.** Undermining.
33. **C.** Ankle–brachial index testing.
34. **D.** Granulation.
35. **B.** Inappropriate sizing of compression stocking.
36. **A.** Hemosiderin deposits.
37. **B.** Hydrocolloid dressing.
38. **C.** Evisceration.
39. **B.** Foam.
40. **D.** Evacuated hematoma.
41. **D.** Diabetic.
42. **B.** Type 2.
43. **A.** Medical device.
44. **D.** Off-loading and callus debridement.
45. **A.** Arterial.
46. **B.** Fungus infection.
47. **C.** Compression wraps.
48. **D.** Epidermolysis bullosa.
49. **A.** Dactylitis.
50. **C.** Unstageable.

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